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# OTORHINOLARYNGOLOGIC PROBLEMS IN THE MOTHER AND THE NEWBORN INFANT

FRANCIS L. LEDERER, M.D.
CHICAGO, ILL.

The anatomic areas of concern under this heading present interesting conditions in the mother and the newborn infant. In the latter, genetic and nongenetic causes (heredity and environment), account for numerous congenital deficiences and abnormalities which initially present problems of management. While the importance of the mendelian pattern and consanguinity are not disputed, the infectious, chemical, endocrinologic, mechanical, actinic or physical, and nutritional factors have been shown to play a prominent role in the etiology of congenital malformations. Since Gregg reported in the Transactions of the Australian Ophthalmological Society in 1941 upon the increase in the number of congenital cataracts following an outbreak of rubella in 1939, it has been demonstrated that should a woman develop rubella in the first trimester of her pregnancy, there is a ten to twelve per cent chance that her child may be born with a severe hearing or other defect. The most satisfactory approach to the problem at present seems to be as follows: a woman in early pregnancy with no previous history of rubella who has known exposure to the disease should receive immune substance early and in large amount. In most parts of the world, the antibody-containing blood product readily available would be gamma globulin: dosage, 20 ml intramus-

From the Department of Otolaryngology, University of Illinois College of Medicine, Chicago.

cularly. Convalescent-phase plasma or convalescent phase gamma globulin are preferred to ordinary gamma globulin . . . approximate dosage, 10 ml intramuscularly.

The advisability of therapeutic abortion in women with clinical rubella confronts the physician with a difficult problem. It would seem that the most likely candidate for therapeutic abortion is the woman in the earliest weeks of pregnancy (before the fourth week) because it is in this period that the fetus is most susceptible to damage from the infection.

It is still advisable to suggest that young girls should be exposed to rubella and thus provide durable immunity throughout their childbearing years. But still open for development is safe active immunization against rubella.

### THE EAR

*In the Mother.* The emphasis has always been upon the unfavorable effect of pregnancy upon the patient who carries the diagnosis of otosclerosis, a conductive type of hearing loss where, typically, newly formed bony placques characteristically cause the footplate of the stapes to be fixed. It has been argued that the bony alteration has been increased by the calcium demands and recorded history has it that this was accepted as a valid indication for therapeutic abortion. That otosclerosis is made worse by pregnancy, and that a woman with this type of hearing loss should be advised against further pregnancies, is emphatically contested by many otologists. Even the hereditary aspects of the disease has been attacked and de-emphasized. Surveys of routinely sectioned temporal bones have disclosed a high incidence (13 to 30 per cent) of so called "silent" areas of otosclerosis in the normal population and not associated with clinical deafness. This is at least ten times as high as the incidence of clinical otosclerosis. Therefore, this specific incidence of immature foci of otosclerotic bone is not a genetically linked trait. It is true that there is a site of predilection at the *fissula ante fenestra.* Involvement at this area seems to show a family tendency in about 40 per cent of the cases. Because of the progress of surgery in the last 20 years, the important progress in the area of genetics and because of the possibilities that research may lead to a medical approach to correct this faulty bone metabolism, it is possible that in the next generation this problem will be a negligible one. Some clinicians feel that there is no reason why any family with a history of otosclerosis should have concern for future generations.

Abortion is not justified because 1) the effect of pregnancy on otosclerosis is extremely variable and unpredictable; 2) there is no exact relationship between the two conditions. The effect of previous pregnancies is not an accurate index of the possible effect of subsequent ones. 3) The favorable effect of abortion on otosclerosis is likewise inconstant. The progression of hearing loss with pregnancy may or may not be arrested by abortion. 4) The disease does not endanger the life of the mother. 5) The disability of this type of hearing loss is lessened by the advent of modern hearing aids and the promise of successful surgical treatment by means of the fenestration or the mobilization operation. It must also be borne in mind that hearing loss both during pregnancy or immediately after parturition may be induced by the severe toxemias of pregnancy and by the toxic effects of drugs, particularly quinine (abortifacient) and streptomycin. Such drugs may also influence hearing of the unborn infant.

*In the Infant* The external ear originates from the first branchial groove, whose blind end comes to abut against the wall of the tympanic cavity, separated by the tympanic membrane. The auricle is formed from the coalescence of the six tubercles that develop during the sixth week on the caudal border of the first and second branchial arches. These six hillocks have only a topographic significance, as there is no definite agreement as to the anlage of the several segments of the pinna.

Preauricular sinuses and fistulas are congenital anomalies derived from incomplete closure of the clefts between the primordia of the external ear, either the first groove itself or the colliculi brachialis.

The external, middle and internal ear may present a variety of deficiencies and deformities. The simplest type, but nevertheless a source of considerable parental anguish, is the tendency of the external ear to protrude. Attempts to get the resilient fibro-elastic cartilage to stay back by use of adhesive tape or tight fitting caps is useless. Surgical correction can be instituted at a later date. For all congenital states, management may be divided into three stages. The first stage concerns the parents who are due for an explanation so that blame and feelings of guilt are warded off. The infant so very often suffers from extremes of abandonment or overindulgence and protection. The second stage concerns the preparation of the environment, particularly other children, to receive the child so unfortunate as to have a defect or a disturbance of a part or of a special function. The third stage is the rehabilitative one in which various forms of plastic and reconstructive surgery and educational processes are employed.

Malformations and congenital deficiencies of the ear are classified as follows:

1. Variations in size and contour of the external ear include familial types, macacus (pointed instead of rounded helix), satyr (pointed and retaining a projection known as a darwinian tubercle), drooping, roll or dog ear, cleft lobule, abnormal protrusion, complete absence of one or both (anotia), absence of a part, asymmetry, microtia, macrotia, polyotia, preauricular fistula, exostoses, and complete or partial stenosis of the external auditory meatus or canal or both.

2. Abnormalities of the tympanic membrane, tensor tympani and stapedius muscles, the ossicular chain (avian or columellar form, or deformity or even fixation of the stapes), the eustachian tube, the facial nerve, and other malformations of the temporal bone and skull. Dehiscences in the course of the facial nerve may occur. Of importance to the obstetrician is the superficial position of the nerve as it emerges from the stylomastod foramen.

3. Aplasia and hypoplasia of internal ear structures including the organ of Corti and the components of the eighth cranial nerve.

4. Familial deafness in association with blue sclerae, and fragilitas ossium (Adair-Dighton's syndrome).

5. Tumors of the ear, including lymphangiomas, hemangiomas, papillomas, cysts, epidermoids, and dermoids. Cholesteatoma may be encountered in various bones of the skull.

Impaired hearing dating from birth may be endogenous, in which case it is due to a malformation or aplasia of the cochlear structures due to an inherited recessive characteristic (mendelian-like incidence); or it may be exogenous, the result of a virus infection in the mother during pregnancy, particularly rubella during the first trimester; or to drug intoxication in the mother during pregnancy, particularly from quinine, intrauterine accidents, or to a birth injury with damage to the cerebral hearing centers.

Prior to 1941 rubella was considered to be a benign and unimportant disease of childhood. During the past two decades it has been repeatedly indicted as a prominent cause of congenital malformations in the newborn. In the first three months of gestation, rubella may result in what has been called the "rubella syndrome" or rubeollar embryopathy, including congenital cataracts, deafness, microcephaly, mental retardation, heart disease (such as patent ductus arteriosus), and a general stunting of growth and development of the child.

Spontaneous abortions and stillbirths have also been attributed to maternal rubella infection. Recent reports indicate that there is a 10 to 12 per cent incidence of congenital malformations in the first trimester and that thereafter rubeollar infections in the mother are not likely to cause damage.

There are a large number of instances of perceptive deafness of obscure origin. Since attention was focused on the relation of maternal rubella and deafness, it was hoped that with this knowledge the number of these cases might be noticeably reduced. It has now been shown that some of these cases are associated with severe neonatal jaundice, usually, but not invariably due to Rh isoimmunization. This type of deafness differs pathologically from that associated with maternal rubella, for the former interferes with the normal development of the primitive cells forming the organ of Corti, whereas in deafness associated with kernicterus, the organs of hearing develop normally but the cochlear nuclei are damaged. Perceptive deafness, usually of a high tone type, is commonly observed in kernicterus. The profundity of the hearing loss bears no relation to the severity of the other disabilities. All patients with kernicterus should be examined by pure tone audiometry. Assessment of intelligence in cases of kernicterus should be made in the light of the otological findings, and the type of intelligence tests selected accordingly.

Erythroblastosis fetalis is a hemolytic disease of fetal or neonatal life due to maternal iso-hemoaglutinin in serum. Three syndromes, viz., congenital anemia of the newborn, icterus gravis, and hydrops fetalis are frequently associated with the development of deafness (an estimated 20 to 30 per cent of those infants who survive). The prognosis for the outcome of pregnancy must be guarded. Mortality rates without treatment are about 65 per cent. Until the infant is born, there is no way at present of determining how severely affected the hearing will be. It is not the Rh factor alone which can produce kernicteric and cochlear nuclear lesions. The icteric lesion can be the sequel of isoimmunization by other serologic incompatibilities. However, the icteric factor alone does not produce the lesions but rather a cellular anoxia which is produced by the deposition of pigment. Some observers view these children as primarily brain-injured, and hold that either aphasia or deafness, or both, may be present. Also, that other conditions such as dyslexia, dysgraphia, agnosia, and apraxia should be considered, inasmuch as all of these conditions occur in brain-injured children and affect auditory behavior and language development. When antibodies appear for the first time late in pregnancy, the infant is usually normal or only slightly affected. The

first erythroblastotic child delivered by an immunized woman usually survives. The prognosis, is however, poorer in subsequent pregnancies, and mortality rates are highest among children of women who have had a previous death or stillbirth because of erythroblastosis. Even these patients, however, may occasionally deliver a mildly affected surviving child.

In recent years, cerebral hypoxia and anoxia, with or without icterus, in the newborn, and prematurity, have become associated in our experience with hearing loss. In the latter group a number of variables are recognized, viz., length of gestation, birth weight, postnatal chemotherapeutic management, duration of incubation and the concentration of oxygen during this period. It has been ascertained that twenty per cent of the premature population of six years of age have some degree of perceptive hearing loss from moderate higher frequency losses to educationally handicapping severe forms. This is five to six times the normal population percentage, after eliminating all patients with a history of or evidence of middle ear pathology. In other studies, mental scores tend to be lower among less premature children, although this tendency is not statistically significant.

Modern technique of discovering hearing loss in the newborn should be known. While the neonatal history is important, the behavioral patterns of the baby are also important. The "very good" and "soft" crying child needs to be appraised by producing a noise and observing of the aural-palpebral reflex. Noise makers which do not induce the kinesthetic response are used. The use of a psychogalvanometric skin resistance (PGSR) has been widely employed, as well as electro-encephalography in the sleeping infant.

Later, the child can be tested for his ability to hear pure tones (tuning forks and audiometry) and speech reception tests in addition to the social, psychological and educational evaluation. This takes an integrated effort of medical and resource personnel, educators and acousticians. Four main directions of thinking prevail: 1) parental education; 2) proper diagnosis and estimation of residual hearing; 3) education by means of auditory training, language training, speech reading, and the use of amplified sounds; and 4) an adequate follow-up program.

Of prime importance in the development of the child with hearing loss is the attitude of the parent toward the child and his problems. While many parents are able to give the child the love and understanding, the discovery of a defect sometimes brings on certain

negative feelings. Guilt feelings at producing a child with such a problem cause a parent to reject the child overtly or to become oversolicitous and demand unreasonably perfect performance, only dimly masking the true feelings underneath, feelings of which the child is all too often aware. In such instances, the child is the living example which continually reminds the parents of their child's problems and makes them struggle unconsciously with the feelings of rejection toward the child. Not infrequently, the father blames the mother and the mother blames herself, placing the child in the midst of a constant stream of tension flowing between parents and between the parents and the child. Then, too, there are parents who compound the problem by clinging to the hope that hearing can be restored by medical or magical means. They plead for fenestration operations, spines are adjusted, airplane rides are suggested, adenoids are removed, all usually with little or no effect upon hearing. Slightly more realistic parents may accept the handicap but expect use of a hearing aid (now being fitted at the age of two years) to endow the child with perfect hearing, normal speech and language, and no observable handicap. The truth is that in most instances where there is severe loss of hearing, there will always be a handicap. Each parent needs to work out his own adjustment to his child and his problem. The well adjusted parent aims to help this child become a competent, happy, well adjusted, hearing-impaired person who communicates with relative ease and is comfortable in the hearing world, rather than a poor imitation of a hearing person, always laboring to conceal his handicap and never succeeding.

### THE NOSE AND SINUSES

*In the Mother.* The nose responds to emotional stress, endocrinological alterations, and drugs, by a vasomotor manifestation characterized by a boggy swelling of turbinates, an increase in the amount of secretion, sneezing and headaches which result from obstruction (usually alternating one side, then the other) of the meatuses leading to the various sinuses. Superimposed suppuration as a result of upper respiratory tract infections may occur. Of course, any condition affecting the adult nose may call for diagnostic and therapeutic aid. Among them is the problem of nose bleeds. Anxiety patterns frequently follow the show of blood from the nose. Identification of a site at the anterior portion of the nasal septum (locus of Little-Kiesselbach) is readily traumatized by the vigor of cleansing the nose digitally (epistaxis digitorum) or by the pattern of the kerchief on the forefinger (epistaxis feminis) which is so often merely a nervous habit. After reassurance, based upon careful examination

to rule out the infrequent presence of multiple small angiomas on the mucosal surface (Rendu-Weber-Osler-Babington disease; familial or hereditary angiomatosis), mucous membrane changes due to various forms of rhinitis or to general and constitutitnal states, the effective treatment of epistaxis requires a determination of the exact cause and site of bleeding.

*In the Infant.* Certain developmental disturbances, infantile bronchitis and laryngitis are caused by nasal insufficiency. Adenoid vegetations are a prominent cause of difficulty. The nose is a starting point for many infections such as: aural, dental (deciduous teeth), ocular, endocranial, pharyngeal, sinal, gastrointestinal, renal, laryngeal, bronchopulmonary, lymphatic and blood stream. The younger the child the greater the susceptibility to infection and its spread to eustachian tube, middle ear, sinuses and tracheobronchial passages. Colds, bronchitis, and otitis media are more common in the first three months of life than during the period of dentition. Once an infant has bronchitis he shows greater susceptibility than others who have been free from it. Those mothers who have nasal infections frequently have infants whose dentition is accompanied by colds, bronchitis and otitis media. The latter, because of the more patent and straighter eustachian tube permit ready access to the middle ear by secretion and even fluids in the process of regurgitation.

Malformations and deficiencies of the nose and sinuses take many forms during the complicated developmental processes. Maldevelopment may vary from slight deformity to some grotesque formation. While the incidence of nasal malformations are not as frequent as those of the ear, abnormalities do result from incomplete union of two or more of the processes participating in the formation of the face. In some instances they are due to a malposition or maldevelopment of these parts. The endogenous and exogenous factors that play a role in the genesis of such maldevelopments are interesting. They may be related to the same factors discussed under congenital ear deformities. The following classification is offered:

I. Variations in size and contour reflecting racial or familial types which result in the hump, the broad, the flat, the bulbous, the beaked, the wide, the flaring alae and numerous other nasal shapes.

II. Agenesis and Imperforation

1. Proboscis lateralis, where one half of the nose is normally formed, the other half being represented by a short, sessile structure attached at the inner canthus of the eye.

There is a coloboma of the eyelid, while the nasal bone, the nasal cavity and the intermaxillary bone on the deformed side are absent.

2. The division of single autositic monsters, often designated as cyclops and synopsia, is characterized by greater or lesser agenesis of the olfactory organs and imperfect development of the visual apparatus. Designations of varieties are ethmocephalus, cebocephalus, rhinocephalus, cyclocephalus and stomacephalus. Arhinencephaly (Kundrat's cebocephalic form), where a rudimentary nose with a single nostril is formed. The structures which may be absent are the horizontal and vertical plate of the ethmoid, the lesser wings of the sphenoid, the optic foramen, the vomer, the lacrimal and intermaxillary bones. The frontal lobes of the brain may be fused in the midline, with maldevelopment of the olfactory area and other parts of the brain.

3. Acrocephalosyndactylism or acrosphenodactylism (Apert's syndrome), where there is pointed shape to the top of the head, hyperteliorism and syndactylia of all four extremities; also obesity, hypogenitalism, retinitis pigmentosa, skull defects and syndactylism (Lawrence-Moon-Biedl syndrome); craniofacial dysostosis (Crouson's s.).

4. Atresia

A. Anterior nares usually consists of a web of skin partially or completely occluding one or both nostrils.

B. Posterior choanae, one or both may be partially or completely occluding one or both posterior choanae. It may be osseous in character (due to an anomaly of the internal pterygoid plate of the sphenoid bone) or membranous, a result of the failure of the primitive buccopharyngeal membrane to rupture (persistence of nasobuccal membrane) or to secondary epithelial overgrowths or plugs.

III. Lack of fusion

1. Median nasal cleft

2. Cleft lip

3. Cleft palate

4. Hypertelorism (abnormal width between the eyes)

IV. Cysts and fistulous tracts

V. Cerebral herniations
   1. Encephalocele
   2. Meningocele
   3. Encephalomyelocele

VI. Septal deviation and subluxation

VII. Osteomyelitis of the maxilla

The fact that congenital deformities occur in multiples in the same subject has long been recognized. In 1900, E. Treacher Collins described a syndrome which characteristically may have combinations of a beaked nasal deformity, bilateral notchings of the lower eyelids, deficiency of the malar bone and infraorbital ridge, lack of cilia on medial third of lower lids associated with oblique palpebral fissures, micrognathia, absence of puncta of lower lids, bilateral external ear deformities, absence of external auditory canal, malocclusion, cleft lip and palate, choanal atresia, and deformities of the extremities. Pierre-Robins syndrome has a few of the involvements mentioned, including micrognathia, cleft palate and cleft lip. A syndrome of importance bears the name Kartagener, in which there is a situs inversus associated with suppurative sinus disease and bronchiectasis.

Less dramatic but more frequent, is the problem related to septal deviations in infants. Numerous theories have been advanced concerning their etiology but no one has been generally accepted. Much evidence has been brought to bear to indicate that there is some inherent developmental factor present. Findings on newborn cleft palate infants has revealed deviations that surely could not be ascribed to pressure of the surrounding structural framework or to trauma. Some observers contend that septal deformities are determined by the genes.

Atresia of the posterior choanae is not generally known as a cause of asphyxia neonatorum, or is it usually associated with fatal suffocation of the infant. Many newborn babies undoubtedly die from suffocation because the atresia was not recognized. It is natural for an infant to breathe only through its nose. If an infant cannot breathe through its nose, it will not open its mouth to breathe. This can be demonstrated in the nursery by pinching the nostrils of a newborn infant when it is resting quietly. The infant in its natural

attempt at forced nasal breathing will not open its mouth to breathe, but instead, will close it tighter by sucking in its lips, completely cutting off its only source of air to maintain life. The infant will then become red in the face, toss its head from side to side and manifest signs of impending suffocation, until nasal breathing is again restored. This reaction does not occur with all infants, probably because some are mouth breathers from the outset and may account for survival of some infants with bilateral choanal atresia surviving.

There is no reflex present to cause an infant to open its mouth to breathe, if it cannot breathe through its nose. As a matter of fact, the infant's natural reflex is to close its mouth tighter in the normal attempt at forced nasal breathing. Opening the mouth to breathe is always a voluntary action. An infant lacks this control, and, therefore, does not open its mouth. Mouth breathing is never normal at any time, or at any age. To prevent suffocation of the newborn, the immediate establishment of an adequate airway is essential.

Many infants die at birth from atelectasis as the primary cause, with no definite explanation for its existence, except to term it congenital. Although bilateral atresia of the posterior choanae may result in death of an infant, only atelectasis will be demonstrated at autopsy. Prompt diagnosis and treatment would have prevented such a termination. In unilateral atresia, accidental closure of the patent nostril will create the same problem as bilateral atresia, and unless corrected, difficulty will be encountered.

The embryonic period, which lasts to the end of the second month, witnesses the differentiation of the main features of the nasal cavities. At the end of the embryonic period the lateral wall of the nasal cavities presents three main turbinates; but the primordia of the sinuses are not yet established. The paranasal sinuses arise as localized epithelial sprouts or recesses of the nasal mucoa during the fetal period of prenatal development which is primarily a period of growth of previously differentiated primordia of organs. The mucosal recesses leading to the establishment of the maxillary and the sphenoid sinuses arise during the third fetal month. The epithelial sprouts of the ethmoid cells are established during the fifth and sixth prenatal month. Until birth these mucosal recesses remain small and do not become "sinuses" until they pneumatize the bones after which they are named, essentially a postnatal process. In the first year of life the maxilla is occupied mainly by a dental sac. The expansion of the sinus occurs at the rate of 2 to 3 mm each year. The first dentition exercises no influence on the direction of growth or on the form of the maxillary

sinus. The second dentition, particularly the germs of the permanent molars and the canine teeth contribute to the molding of the floor of the expanding maxillary sinus. Occasional variations exist in the size and shape of the maxillary sinuses but not as many as in the development of the various ethmoid cells. At birth these groups are fairly well formed and the height of the ethmoid is twice that of the maxillary portion. The ethmoid cells develop relatively rapidly.

## THE ORAL CAVITY

*In the Mother.* The pharynx may be the site of inflammatory conditions as it is under ordinary circumstances. Mechanical, chemical, bacteriologic and systemic causes may play a role. Pregnancy may be regarded as either causative or predisposing to a stomatitis. Abstinence from irritating foods, maintenance of good general health and special attention to correct mouth hygiene are paramount. Elimination of all sources of mechanical, thermic or chemical irritation is paramount. The need is frequently for a good basic knowledge of mucous membrane lesions, the ability to examine the areas skillfully, so that reassurance of the patient concerned about signs and symptoms may be on firm and reliable grounds. Proper mouth and dental hygiene in the prospective mother needs to be constantly stressed.

Pregnancy tumor (*gingivitis gravidarum*) is an alteration of the gingival margin, various types of which may occur during pregnancy. It consists essentially of granulation tissue similar in structure to a pyogenic granuloma. The typical pregnancy granuloma is a lobulated mass covered by hyperplastic epithelium which may become ulcerated.

Ulcerative and membranous lesions of the mouth are always challenging diagnostic problems. They may vary from a simple herpetic lesion to a recurrent type of aphthosis which may have similar lesions in the vaginal vault (Behcet's syndrome). The mucous membrane may also reflect toxic manifestations of some drug or even a blood disturbance.

*In the Infant.* The pharynx becomes modified many times during the course of its evolution. Many of the derivatives appear early in the phylogenesis and migrate considerable distances from their origin. The number and complexity of developing parts permit the possible occurrence of numerous anomalies.

The oral cavity begins as an ectodermal depression, the stomodeum, on the ventral surface of the head region. During the fourth

embryonic week, the ectoderm of the stomodeum rapidly approaches the entoderm of the foregut and the two approximate in the oral plate. When the oral plate ruptures, the cephalic opening to the gut is established. This corresponds to the tonsillar region. Three middorsal diverticula appear early in this region. 1) Seessel's pouch or pocket is a small diverticulum of the foregut which extends a short distance cephalic to the oral plate opening. 2) The pharyngeal bursa, a small cleft which frequently persists in the posterior wall of the future nasopharynx. 3) Rathke's pouch or pocket, an ectodermal diverticulum which arises middorsally from the stomodeum and later fuses with the hypophysis to become part of the pituitary gland, occasionally remaining patent in the adenoid region.

The stomodeum, at first shallow, is deepened by the forward growth of the surrounding structures which give rise to the face and jaws. The prominent mandibular arch forms the caudal boundary and the frontal process the cephalic boundary of the oral cavity. In the fifth week the maxillary processes grow in from the angles of the cavity. Shelf-like outgrowths arise from these processes, grow medially and fuse in the midline to form the palate, thus dividing the stomodeum into two parts, the oral and the nasal cavity. The nasomedial processes complete the palate by forming the small premaxilla.

The lips are formed when an ingrowth of epithelium at the periphery of the oral fissure splits to form the gingivolabial groove, separating the lips from the developing gingival and teeth, the formation of the latter beginning with a band of epithelium conforming to the arch of the jaws, condensing and growing into the gingival mesenchyme. This band, the dental lamina at intervals forms condensations which take the form of inverted cups, the enamel organs. The ameloblast cells lining the cups are destined to form the hard enamel of the teeth. Proliferating bone on the margin of the jaws sends forth alveolar processes which surround the teeth.

The tongue is a mucous membrane sac which becomes filled with a mass of developing muscles. The tongue consists of two portions, the body anteriorly and the base posteriorly. The anterior portion is formed from the region between the first and second branchial arch from which the tuberculum impar projects dorsally. Behind the tuberculum is a deep evagination of epithelium, the median thyroid evagination, and behind this is a transverse elevation formed by the ventral ends of the second and third branchial arches. The foramen caecum is a vestige of the entodermal invagination of the thyroid primordium and lies in the ventral surface of the anterior extremity

of the foregut. This is a landmark forming the dividing line between the ectodermal and entodermal mucosa covering the tongue.

The pharynx is divided into three divisions; that above the level of the posterior margin of the soft palate being the epi- or naso-pharynx, containing the eustachian tube orifies and that part of the lymphoid ring (Waldeyer) known as the adenoids; the oro- or meso-pharynx which lies between the soft palate and the upper border of the epiglottis, containing the faucial tonsil and the lingual tonsil at the base of the tongue, and the laryngo- or hypopharynx, which extends from the tip of the epiglottis to the lower border of the cricoid cartilage, where the epiglottis begins.

The classification of deformities and anomalies about the mouth:

I. Lips and Face
   1. Double lip
   2. Macrocheilia
   3. Clefts
      A. Straight, of lip and check
      B. Oblique, of lip and cheek
      C. Lateral facial (maxillo-mandibular)
      D. Partial or complete of lip (with or without palatal cleft) unilateral or bilateral
   4. Fistulas of lower lip
   5. Elongated and hypertrophic frenum

II. The Jaws
   1. Mandible
      A. Prognathia
         1. Entire
         2. Partial, of alveolar process (prophatnia)
      B. Micrognathia
   2. Maxilla
      A. Abnormal protrusion
      B. Scaphoid Face

C. Multifacial Dystrophy

D. Hemidystrophy

3. Alveolar Process

A. Cleft

B. Dermoid Cyst

C. Prophatnia

III. Hard Palate

1. Cleft

A. Partial or complete

B. Unilateral or bilateral

2. Short palate

3. Torus palatinus

IV. Soft Palate

1. Bifid uvula

A. Partial

B. Complete

2. Submucous cleft

V. Tongue

1. Ankyloglossia (tongue tie)

2. Bifid tongue

3. Absence of papillae

4. Fissured tongue

5. Macroglossia

6. Thyroglossal duct cyst

7. Lingual thyroid

8. Median rhomboid glossitis

9. Psaumé syndrome

VI. Mesopharynx

1. Branchial fistula and cyst

2. Cleft or absent pillars
3. Cervical synostosis

VII. Nasopharynx

1. Narrowing
2. Shortening
3. Pertik's and Kirchner's diverticulum
4. Choanal atresia
5. Meningocele and meningoencephalocele
6. Inclusion dermoids and teratoid growths

VIII. Floor of Mouth

1. Dermoids and cysts
2. Ankyloglossia
3. Angiomas and lymphangiomas

IX. Salivary Glands

1. Congenital malformations
   A. Absence of one or more pairs
   B. Abnormal fistulous openings
   C. Obliteration of ducts
   D. Cyst formations
   E. Tumors
2. Aberrant glands

The final size and contour of the nasopharynx depend upon the angle between the posterior margin of the vomer and the pars basilaris. The greater this angle, the larger is the nasopharyngeal space. This angle may show variations between 76 and 124 degrees. Prominence of the anterior tubercle of the atlas or epistropheus or both may lead to respiratory diffculty. In infants, the head is large, the vertebral column is soft, and the resulting lordosis may become so pronounced as to markedly narrow the nasopharyngeal space. The space may be narrowed (saggital diameter) by shortening of the base of the skull as occurs in chondrodystrophy due to tribasilar synostosis, congenital dislocation or ossification, and luxation of the atlas. Premature

synostosis of the sutures (oxycephaly) may lead to downward protrusion of the pterygoid process, which in turn produces a shortening of the nasopharynx in the vertical diameter.

The pharyngeal end of the eustachian tube in the newborn is situated slightly below the level of the soft palate. Individual variations in configuration and situation of the ostium are not uncommon. Abnormal width and patency renders the tube even more susceptible to the ingress of fluids and organisms into the middle ear. A recess between the salpingopalatine fold and levator ridge may extend downward from the ostium (recessus salpingopharyngeus). Pertik's diverticulum, arising from Rosenmüller's fossa and extending along the upper margin of the superior pharyngeal muscle and the diverticulum of Kirchner arising from the floor of the eustachian tube near the ostium, extending between the tensor and levator veli palatini muscle, have been encountered.

Failure of obliteration of the branchial cleft may result in a fistula with internal and external openings, one or the other, or both. The fistula passes below the stylopharyngeal muscle and opens at the lateral margins of the neck anterior to the sternomastoid muscle. The internal opening is usually in the pharynx lateral to the posterior tonsillar pillar, below the tonsil or in the hypopharynx, but more frequently below and behind the angle of the jaw. Occasionally it opens into Rosenmüller's fossa. Cysts, containing glairy mucus, are found under the sternocleidomastoid muscle and not usually manifest at birth.

The third and fourth pharyngeal pouches, evaginations and condensations of entoderm, form the basis for the development of the thyroid gland, the thymus, and the lateral thyroid primordia. The thyroid gland with its lobe and isthmus situated in relation to the upper rings of the trachea, frequently has a central or pyramidal lobe connected with the point of origin of the fetal thyroid, the foramen caecum. The connecting anatomic structure is the obliterated thyroglossal duct, anywhere along which tract aberrant thyroid tissue is found but is more frequently encountered at the base of the tongue. It is then designated lingual thyroid. If the upper part of the duct remains unobliterated a dermoid cyst may develop, and when the lower part is patent, a cyst appears, generally situated in the midline of the neck. About the fourth week of the embryo there appears in the midline, immediately posterior to *tuberculum impar,* a diverticulum, which at first is hollow but later becomes solid. This diverticulum grows downward and eventually becomes the thyroid gland,

the upper extremely forming the foramen caecum. The patency of the central duct which descends from the foramen caecum results in the thyroglossal duct which runs downward behind the hyoid bone and terminates in the deep fascia in the region of the thyroid isthmus.

In typical palatal incompetence or short palate, several clinical signs are observed that lead to the suspicion this type of congenital abnormality. The presence of a large antero-posterior diameter of the nasopharynx, the absence of a median raphe, the presence of a bifid uvula and the veil-like appearance of the soft palate should lead the clinician to suspect such a possibility. A subsequent tonsillectomy and adenoidectomy may bring to light the incompetence of such a palate and prove extremely detrimental to the speech of such an individual.

In a Pierre-Robin syndrome, a marked micrognathia is found to be associated with either an obvious cleft of the palate or a submucous cleft which appears as a pit in the midline. It is the micrognathia which results in a retropositioning of the tongue so as to encroach on the airway, leading to respiratory obstruction necessitating tracheotomy.

The classification of clefts of the palate is largely determined by the anatomic categories and these are grouped according to:

1. Posterior clefts which include those clefts solely of the soft palate, as well as clefts of the soft palate and a portion of the hard palate.

2. Unilateral clefts are those of the lip and palate which occur on either side of the face and are involved in a fusion of one of the palatine processes with the inferior border of the vomer.

3. Bilateral clefts are those in which the lip and palate are involved and the premaxillary segment is completely separated from the lateral palatine processes.

4. The alveolar clefts are defined as clefts of varying degree of the alveolar process and are usually associated with a cleft lip.

Bednar's aphthae is a disease of marasmic infants, localized near the posterior third of the hard palate, close to its junction with the soft palate. The characteristic lesions are small round ulcers in the mucous membrane with non-infiltrated borders. These lesions are situated near the midline or over the pterygoid area, spreading forward to the raphe of the hard palate in butterfly formation. There is a

peculiar condition of the mouth of the newborn which must be differentiated from Bednar's aphthae. Round, firm nodules of pinhead size are found in the oral mucosa during the first weeks of life. They have been designated as epithelial pearls. Occasionally those over the raphe of the hard palate undergo erosion and slight ulceration.

A mycotic form of stomatitis, known as "thrush," "white mouth or sore," is due to candida albicans or monilia albicans. It is observed in infants born to mothers suffering from vaginal candidiasis, contamination of the mouth of the newborn taking place as the head passes through the birth canal. Thrush produces whitish spots, especially on the tip of the tongue. These areas may become enlarged through coalescence and consist of leukocytes, epithelial cells, fibrin, mycelium and spores. Such areas may be wiped away without causing bleeding. This is contrary to the intensely hyperemic appearance of mucosa occurring in neisserian contamination of the mouths of infants who become infected during birth.

### THE LARYNX

*In the Mother.* Most forms of laryngeal disease have many symptoms in common which are due to interference with the essential functions of phonation, respiration and deglutition. When the position of the larynx in the structural framework of the neck, its lymphatic and nerve supply are considered, it is comparatively simple to visualize the multitudinous variety of acute and chronic inflammations, disturbances of innervation, and pathologic alterations as the result of external as well as local and systemic factors that are capable of affecting this organ. External irritants, to say nothing of smoking, vocal abuse and diseases of the upper and lower respiratory tract account for many of the cases wherein hoarseness is the main symptom. The emotional impact of pregnancy, particularly in the first trimester, is not infrequently accompanied by partial or total aphonia. It is apparent that when a wide divergence of possibilities exist to cause a disturbance in the voice, it is of utmost importance that the physician be able to visualize the larynx and vocal cords by means of a laryngeal mirror. This would remove all guesswork and permit early and adequate rational therapy.

*In the Infant.* The larynx in the normal infant is situated at the level of the fifth cervical vertebra. Its descent is gradual. The entrance to the larynx is at an angle from behind forward and downward toward the glottic lumen. With descent of the larynx as the infant grows, the V-shaped (narrow and folded longitudinally) epiglottis

assumes a more nearly vertical position, placing the axis of the lumen more nearly in a line with the subglottic larynx and trachea. The glottis is 7 to 9 mm in its anteroposterior measurement and the subglottic larynx 1 or 2 mm less in diameter (about 6 mm). The tracheal diameter is several millimeters larger than the subglottic larynx. The infant larynx and its framework are softer and more pliable than in the adult. In addition to these anatomical variations, it should be noted that the infant's nervous system is relatively irritable and unstable, therefore readily thrown into spasm by the slightest stimulation of the respiratory or digestive systems. Hyperexcitability of the neuromuscular apparatus of the larynx is an important factor in the production of stridor.

Congenital anomalies of the larynx consist of membranes (webs), absence of tracheal or laryngeal cartilages and an exaggerated type of infantile or fetal larynx producing a stenosis at birth. They are frequently associated with other congenital anomalies and malformations. The following classification is suggested:

I. Malformations
   1. Membrane (web)
   2. Absence of cartilages
   3. Infantile (exaggerated type)
   4. Epiglottis
      A. Deformed
      B. Flaccid
      C. Cyst
   5. Mandibular hyper- and hypoplasia
   6. Macroglossia

II. Tumors
   1. Laryngeal (papilloma)
   2. Mediastinal
   3. Cysts

III. Enlarged thyroid or thymus

IV. Laryngomalacia

V. Paralysis of cords
   1. Injuries
   2. Erbs'
   3. Cardiac
   4. Vascular
   5. Central
      A. Meningocele
      B. Cerebral agenesis

Stridor in infants suggests pharyngeal, laryngeal or tracheal disease or lesions causing pressure on these structures. The condition may or may not be serious, but its cause should be determined. The history and clinical study of the type and phase of the stridor will suggest a tentative diagnosis, but radiologic, laryngoscopic, bronchoscopic or esophagoscopic examination must be made to provide the final estimate of the true nature of the lesion. In inflammatory processes and in trauma the stridor is characterized by an acute onset. In congenital lesions the history generally indicates that symptoms have been present since birth. Stridor during the first months of life is most often due to a congenitally soft, flaccid larynx, although congenital cysts, webs, paralyses of the larynx, and congenital vascular anomalies compressing the trachea will give similar signs and symptoms. Inasmuch as the latter are frequently serious, an accurate differential diagnosis is imperative.

### THE LOWER RESPIRATORY TRACT

*In the Mother.* The trachea and bronchi act so obviously as air conduits to the lungs that they are commonly thought of as purely passive organs, subservient to the respiratory elements of the lung. Less obvious is the fact that in addition to this passive function they have active properties of a protective nature against bacterial and foreign body invaders. The trachea only attracts attention when involved in obstructive processes and the bronchi are dealt with when there is a bronchiolar or parenchymal change. Many pulmonic changes occur when the trachea and bronchi have not been effective barriers to infection. A careful history, physical examination, roentgenographic studies and other laboratory aids, and skilled bronchoscopic technique serve to ascertain causes of cough, secretion, obstruction, or blood and make possible the therapy to combat such signs and symptoms.

*In the Infant.* The normal development of the trachea and lungs proceeds from two major constituent parts. The entodermal primordium arises as a ventral outgrowth from the primitive foregut at three and one-half weeks when the embryo measures but a few millimeters in length. It forms the epithelial lining of the entire tracheobronchial tree and the glandular derivatives. It constitutes the mold for the developing lower respiratory tract, determining the bronchi and all of their subdivisions. This entodermal primordium becomes surrounded by the mesodermal constituent part which forms the connective tissue, septa, smooth muscle, cartilaginous plates, the vascular bed and the visceral pleura.

At seven weeks of development the processes of secondary and tertiary subdivision of the bronchi and that of lobation in the total lung primordia are well under way. The further development of the bronchial tree progresses rapidly through the end of the second month and early in the third month. The uninflated lungs assume more and more the form of a multi-lobed glandular primordium. Tracheal and bronchial cartilages become established at this time. Also at this time, the pleural cavities are definitely separated from the peritoneal cavity and the heart, diaphragm and liver exert secondary formative influences upon the expanding lungs.

During the developmental phase extending from the fifth to the tenth week of embryonic life, the lungs are provided with temporary supernumerary lobes. This results from the development of dorsal buds from the stem bronchi of both lungs and of the primordium for an infracardiac lobe of the right lung. Such additional lobes usually become fused with the inferior lobes, but may persist to form accessory or anomalous pulmonary lobes. The major fissures which at first are nearly transverse assume oblique positions with the subsequent expansion of the lungs. At seven and one-half weeks (20 mm embryo) the superior lobes expand craniad to form definite pulmonary apices. Simultaneously, the shaping influences of the enlarging liver and of the diaphragm establish the flattened bases of the lungs.

The definite general form of the lungs usually is attained early in the third month of intrauterine development (3 cm fetus). It is obvious, therefore, that truly abnormal development of the tracheopulmonary primordium must occur before that time. During the embryonic period, the potentiality for branching is greatest in the bronchial tree. Supernumerary and subnumerary lobes will then arise or normal growth most readily disturbed. During the later fetal life, other anomalous lung conditions may arise, e.g., congenital cysts,

atelectasis, etc., but they are of a pathologic rather than of a teratologic nature.

A classification of the anomalies of the lower respiratory tract, modified by Holinger, is as follows:

I. Anomalies of the trachea
   1. Agenesis and atresia
   2. Constriction and enlargements
      A. Cartilaginous deformity
      B. Anomalous vascular compression
      C. Webs
      D. Fibrous stenosis
      E. Tracheomalacia
   3. Evaginations and outgrowths
      A. Tracheocele, diverticula and cyst
      B. Fistulas
      C. Tracheal lung
   4. Abnormal bifurcation or deviation
   5. Other anomalies of gross morphology

II. Anomalies of the bronchi and lungs
   1. Agenesis and atresia
   2. Constrictions and enlargements
      A. Webs
      B. Compression from cardiovascular anomalies
   3. Evaginations or outgrowths
      A. Bronchoceles, diverticula
      B. Cysts and emphysematous lobes
      C. Fistulas
   4. Subnumerary bronchi, lobes and fissures
   5. Supernumerary bronchi, lobes and fissures

6. Anomalous bronchial and lung tissue attached to the respiratory system

7. Anomalous bronchial or lung tissue attached to tissues other than respiratory system

8. Kartagener's syndrome

The diagnosis of congenital malformations of the lower respiratory tract during the life of an infant is made with the help of team work by the pediatrician, roentgenologist, bronchologist and thoracic surgeon. The history of obstetrical trauma or subsequent chest injury is important. From the physical findings a differentiation may be made of laryngeal anomaly from other types of obstruction to respiration by the character of the cry and by direct laryngoscopy. Physical examination of the chest is essential in the correct interpretation of the x-ray film. For example, differentiation must be made between an obstructive emphysema of one lung with compression atelectasis of the opposite lung and an obstructive atelectasis of one lung with compensatory emphysema of the opposite lung. An erroneous interpretation may result if reliance is placed on a single chest film, the report in the former case stressing the atelectasis of the relatively uninvolved side rather than the much more significant obstructive emphysema of the opposite side. Fluoroscopic study, x-ray films in inspiration and expiration, a lateral view with the arms down and back to reveal the larynx, a barium swallow, bronchography, planography and angiocardiography all add significant information in special instances. Bronchoscopy without anesthesia is now an accepted diagnostic proceduce in unusual respiratory conditions in infants. Limiting the time of the procedure, the use of small instruments and the administration of oxygen through the scope during the examination increase the margin of safety. Finally, exploratory thoracotomy in infants, now a feasible, well established procedure, increases the scope of diagnosis and adds therapeutic possibilities which were previously unavailable.

As in most studies of congenital anomalies, those of the lower respiratory tract have shown frequent associations with those of other organs and systems, as well as a multiplicity of anomalies within the respiratory tract itself.

Congenital anomalies of the bronchi and lungs are relatively common. A large group is comprised of the abnormally developed terminal respiratory units and cyst formation which, when infected, produce clinical manifestations not unlike those of pulmonary sup-

purative lesions, such as bronchiectasis, lung abscess, pneumonitis, and tuberculosis. Congenital bronchiectasis is found in any portion of the lung, most frequently in the left lower lobe. However, symptoms may not become manifest early in life. The condition develops as a result of pressure on a bronchus when medium sized bronchi have been developed, or from an agenesis of the alveoli with enlargement of lung buds as the size of the thorax increases.

Another group consists of large pulmonary cysts which become overdistended due to a check-valve communication with the bronchus. This produces marked alterations in the intrathoracic pressure due to a collapse of a large part of the entire lung. This is observed in the early months of life and is frequently the cause of death. Lung cysts are congenital anomalies which may, however, be entirely compatible with life and are found incidentally at postmortem, or they may be so large as to occupy so much of the thoracic cavity in the newborn that the remaining aerating lung is insufficient for life. They are generally due to agenesis of the alveoli. Occasionally, a ball-valve will develop, permitting air to enter the cyst, but not escape, and death results from compression of the remainder of the chest by the ballooning cyst.

Bronchogenic cysts are considered to be congenital structures formed by the pinching off of a small bud or diverticulum of the foregut. They are frequently found in the posterior mediastinum, near the bifurcation of the trachea. As a rule, they are smooth, ovoid or round, containing a thick, clear, jelly-like fluid. Dermoid cysts and teratomas are usually lobulated and are frequently situated in the anterior mediastinum.

### THE ESOPHAGUS

*In the Mother.* The esophagus is subject to local affections peculiar to its structure, as well as neurogenic and psychogenic, systemic and metastatic states. Infections and malignant neoplasms arising in the neck, thorax and abdomen may spread to the esophagus. Further, the esophagus is subject to direct trauma by ingested corrosive substances or penetrating wounds. Subjective symptoms vary in intensity but may consist of pain and difficulty on swallowing. Symptoms arising from hyperactivity, as reversed peristaltic movements, are frequently observed in psychogenic states. The history, physical examination, roentgenogram, laboratory aids and direct endoscopic observation will establish the diagnosis so rational therapy may be applied.

*In the Infant.* Congenital anomalies of the esophagus result from faulty development at about the fourth week of intrauterine life. A groove in the ventral wall of the primitive pharynx deepens and converts into the laryngotracheal tube. This tube is in intimate relation to the upper part of the foregut, the lengthening of which forms the esophagus. Incomplete separation of the trachea and esophagus and failure of recanalization of the esophagus may result in one type of malformation, such as 1) atresia of the esophagus with tracheo-esophageal fistula, 2) atresia without tracheo-esophageal fistula, or 3) tracheo-esophageal fistula without atresia. Other anomalies encountered are 1) reduplications of the esophagus, 2) para-esophageal cysts, and 3) diverticula. They may be classified as follows:

I. Anomalies of the esophagus
   1. Absence (hypopharynx ends in blind sac; cardia completely closed)
   2. Duplication (a second esophagus may exist)
   3. Atresia (rare without tracheo-esophageal fistula)
   4. Webs (single or multiple)
   5. Stenosis
      A. Fibrous strictures
      B. Failure of epithelization (soft stenosis)
   6. Short esophagus (a portion of cardiac end of stomach above the diaphragm)
   7. Achalasia (cardiospasm)
      A. Simple (failure of neuromuscular mechanism)
      B. Extreme dilatation with fibrous stricture at the cardia

II. Combined esophagotracheal anomalies
   1. Tracheo-esophageal fistula with esophageal atresia
   2. Tracheo-esophageal fistula without esophageal atresia

III. Combined anomalies due to those involving other structures
   1. Obstructions due to cardiovascular malformations
   2. Lung buds of esophageal origin

Atresia of the esophagus should be suspected when a newborn infant drools excessively, chokes upon feedings, and has cyanotic episodes. The diagnosis can be confirmed by the inability to pass a catheter into the stomach and the visualization of a blind upper pouch on roentgenogram after introducing opaque media (*never barium*) into the upper esophagus. The presence of air in the stomach and intestines as evidenced by the roentgenogram establishes the presence of a tracheo-esophageal fistula. Absence of air suggests that there is no fistula between the lower esophagus and trachea, but does not exclude this possibility. Tracheo-esophageal fistula without atresia should be suspected when a newborn infant has severe coughing episodes when fed. The fistula may be demonstrated by roentgenograms obtained after opaque media has been placed in the esophagus with the infant in the prone position. Atresia of the esophagus with tracheo-esophageal fistula in which the lower esophagus enters the posterior wall of the trachea makes up 90 per cent of anomalies of this type.

Atresia of the esophagus without tracheo-esophageal fistula is variable in the length of the upper esophageal pouch. The lower esophagus may vary from an atretic cord of tissue to a sharply tapering esophagus ending blindingly in the posterior mediastinum adherent to, but not communicating, with the trachea. A variant of this type of anomaly is a congenital esophageal web located at the mid-esophagus.

Tracheo-esophageal fistula without atresia is characterized by a well developed esophagus which is intimately adherent to the trachealis muscle along the lower portion of the trachea with the presence of a fistulous opening above the bifurcation. In rare instances, two fistulas may be present, with the upper segment as well as the lower segment communicating with the trachea.

Reduplications of the esophagus and para-esophageal cysts may not be productive of symptoms. Their presence is frequently indicated by an abnormal shadow observed on a roentgenogram of the chest. Surgical removal usually can readily be accomplished by employing a transpleural approach to the posterior mediastinum. Epiphrenic esophageal diverticula are considered to be acquired and of the pulsion type. Congenital anomalies of the esophagus in which atresia or a fistulous connection are present, offer urgent surgical problems. A large percentage of these infants can be salvaged by direct end-to-end anastomosis of the two segments of the esophagus or by delayed substitution procedures when the former is not feasible. Associated anomalies incompatible with life, aspirational episodes

causing pulmonary infections, and complications due to faulty healing of the anastomosed esophagus, are the greatest obstacles encountered in the surgical management of these patients. Other esophageal anomalies present surgical problems less urgent and more readily managed.

## THE NECK

*In the Mother.* The presence of a swelling, especially in the side of the neck, frequently presents a difficult diagnostic problem. The commonest condition is an adenitis associated with an infection in the upper respiratory tract. When such a swelling fails to recede the possibility of an abscess is considered. Absence of fever, leukocytosis and tenderness should lead to suspicion of a tuberculous adenitis. In the absence of inflammatory signs, a branchial cyst should be considered. Firm swellings should suggest metastatic carcinoma, lymphosarcoma, Hodgkin's disease, thyroid enlargements and tumors, leukemia and other blood disturbances. It is apparent that there are difficulties attending a diagnosis of an apparent simple swelling of the neck. It is usually necessary to differentiate between inflammatory (acute or chronic), constitutional and neoplastic diseases. Exhaustive studies should include a careful history, physical findings and laboratory evidence. Aspiration of fluid or for the study of cells and biopsy are important diagnostic aids.

*In the Infant.* In an embryo of three to four weeks, the five branchial arches develop on the lateral surface of the neck. Each arch contains a cartilaginous structure, an aortic vascular arch, muscles and nerves. The arches are separated externally by the branchial grooves (of ectodermal origin), and internally by the pharyngeal pouches (of entodermal origin). The other four arches are the anlage of most of the cervical structures.

The second arch grows rapidly in a downward direction, telescoping the other caudal arches and enclosing the lower branchial grooves in a precervical sinus, which normally becomes obliterated by the fifth or sixth week. If any of the ectodermal elements lining the walls of the precervical sinus become buried in the neck, a tumor, a cyst, or a fistula may develop. The third pharyngeal pouch gives rise to the thymus and inferior parathyroid gland by way of the thymopharyngeal duct. The persistence of fragments of this duct explains the origin of lateral cysts and fistulas of the neck.

Congenital abnormalities of the neck, of interest, are these:

I. Present at birth

1. Misplaced auricle
2. Cysts (hygroma colli, myxoma, atheroma and dermoid)
3. Cavernous angioma, lymphangioma or aneurysm

II. Arising from embryological rests

1. Branchial cyst
2. Thyroglossal duct cyst

Branchial cysts may be divided into four clinical groups: 1) cysts that lie at the anterior border of the sternocleidomastoid muscle; 2) cysts that extend deeper and lie on the great vessel, being often adherent to the internal jugular; 3) cysts that may reach the lateral wall of the pharynx, passing between the external and internal carotid arteries, and 4) cysts that lie on the pharyngeal wall behind the common carotid.

Fistulas may be subdivided into four forms: 1) sinus tract, open externally in the skin, blind internally, as a cul-de-sac; 2) sinus tract, open internally, blind externally, ending as a cul-de-sac; 3) fistulous tract, open internally in the pharynx and externally, and 4) the one which has no external or internal opening, but remains as an unobliterated segment in the neck. The anatomic relations of the fistulas correspond to those of the cysts. In contrast with branchiogenic cysts, sinuses and fistulas present clinical symptoms at birth or in early infancy.

The key points in differentiating the lateral vestigial cysts and fistulas of the neck are based on anatomic and histologic grounds. Cysts and fistulas of branchiogenic origin are derived mainly from the second cleft and pass between the internal and external carotid arteries; the internal opening of a fistula is in the tonsillar fossa. Branchiogenic cysts in close contact with the tail of the parotid may be of first cleft origin. Cysts and fistulas from the thymopharyngeal duct lie anterior and very close to the carotid sheath; the internal opening of a fistula is in the region of the pyriform fossa. Both types of fistula have external openings at the anterior border of the sternocleidomastoid muscle.

The thyroid gland has two anlagen. The lateral thyroid originates from the fourth branchial pouch; the median thyroid is from

a diverticulum in the ventral wall of the pharynx between the tuberculum impar and the copula. Such a diverticulum appears at the end of the fourth week. The thyroid primordium grows caudally and by the fifth week appears as a bilobular structure connected to the pharynx by a stalk, the thyroglossal duct. This pedicle normally disappears by the sixth week, but a pit persists in the midline of the tongue to mark its point of origin, the foramen caecum.

The persistence of epithelial rests in the remnants of the thyroglossal duct results in thyroglossal cysts and fistulas. Abnormalities of the thyroglossal tract appear as cysts or fistulas in the midline of the neck or at one side of the midline from the base of the tongue to the isthmus of the thyroid gland.

The fistulas are congenital and appear as a cord of tissue palpable under the skin without evidence of a cyst. Sinus tracts may develop from a cyst previously lanced or incompletely removed, or one that has become infected. The thyroglossal duct may pass anterior to the hyoid, through the hyoid or, less often, posterior to the hyoid.

Congenital cysts of mesodermic origin in the head and neck are mainly represented by hygroma colli or lymphangioma cystica. Their origin is from a developmental anomaly of the primary lymph spaces. These are multiple thin-walled cysts with colorless or yellow fluid.

1853 West Polk St.

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LXVII

# ULTRASTRUCTURE OF THE TRACHEAL CILIATED MUCOSA IN RAT AND MAN

J. Rhodin, M.D.

New York, N. Y.

New techniques in morphology have been developed during the last eight years. Among these techniques, that of electron microscopy has opened up a new world of details inside a cell, the knowledge of which seems to be of great importance for the understanding of the normal and pathologic activity of cells. The application of the electron microscope in studying the structure of the tracheal epithelium[4] did contribute to the understanding of problems like ciliary activity, mucus secretion, and cell regeneration. The data to be discussed here are taken mainly from this reference. However, some recent experiments have been done with human trachea, generously placed at my disposal by Dr. John F. Daly, Department of Otorhinolaryngology at New York University, Bellevue Medical Center. The micrographs in this communication represent all human trachea, whereas the schematic drawings are the summary of a study of the rat trachea.[4] The tissue is fixed in osmium tetroxide,[2,3,6] dehydrated in alcohols and embedded in liquid plastic[1] which subsequently is made to harden. The specimen can then be sectioned with a special type of microtome which produces sections with a thickness of about one-millionth of an inch.[5] The sections may immediately be examined in the electron microscope without dissolving the embedding medium or staining the tissue. The magnifications used ordinarily range between 2,000 and 20,000 diameters. By subsequent printing, a magnification of 50,000 to 200,000 diameters may easily be achieved, at which structures of molecular size may be pictured.

In the rat, the tracheal epithelium is of a simple columnar type (Fig. 1) whereas in man, it is pseudostratified and columnar. The mucous blanket is formed by secretion from mucous cells as well as serous cells which are encountered both in the epithelial layer and in the subepithelial glands. The continuous beating of the cilia causes

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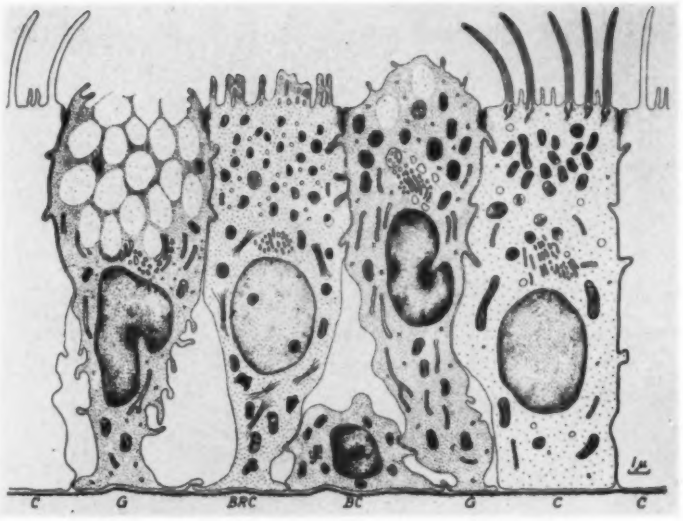


Fig. 1.—A schematic presentation of the tracheal epithelium in the rat. C = ciliated cells; G = mucous cells in different stages of secretion; BRC = brush cell; BC = basal cell. (After Rhodin & Dalhamn, 1956)

the mucous blanket to move upwards. During this movement, the viscosity of the mucus is influenced by a certain evaporation, but additional resorption by the epithelium itself may cause changes of the viscosity.

In the light as well as in the electron microscope, the goblet cells and the ciliated cells are easily recognized. The nuclei are prominent as are the abundant cilia on the surface (Fig. 2).

The mucous granules are formed in the basal part of the goblet cell, and as they enlarge, they approach the luminal part of the cell where but little cytoplasm is left between the fusing mucous granules. The cross section at the lower level of the cell will show the activity of the Golgi complex, the particular structure of cells which is active during secretory processes (Fig. 7). The mucous granules are formed within the Golgi membranes with a probable interaction of the nearby α-cytomembranes. The energy required is supplied by the mitochondria which are known to be the carriers of enzymes in all cells. The density of the granules is very slight due to their content of polysaccharides which do not take stain with osmium tetroxide.

Fig. 2.—Luminal part of human tracheal epithelium with two ciliated (C) and several mucous (M) cells. The cilia (T) are sectioned transversally because of their bent position. Note the abundance of mitochondria in the upper part of the ciliated cell (the dark spherical and elongated structures). The mucous granules are formed in the lower part of the goblet cell. Magnification 4200X.

The next step is the discharge of mucus at the epithelial surface. Apparently, parts of the dense cytoplasmic strands are pulled away from the cell together with the mucous granules. Torn cytoplasmic strands remain connected with the rest of the cytoplasm, and these strands resume the shape of microvilli in the closing luminal gap of the cell. Finally, the crater closes up and we know through information obtained by other sections than those shown here, that the mucous cell now starts the production cycle over again until a new discharge of mucous granules is added to the mucous blanket. This cycle has been demonstrated to be identical in rat and man.

In man, a second type of secretory cell has been found which presumably is responsible for a serous secretion. The shape of the

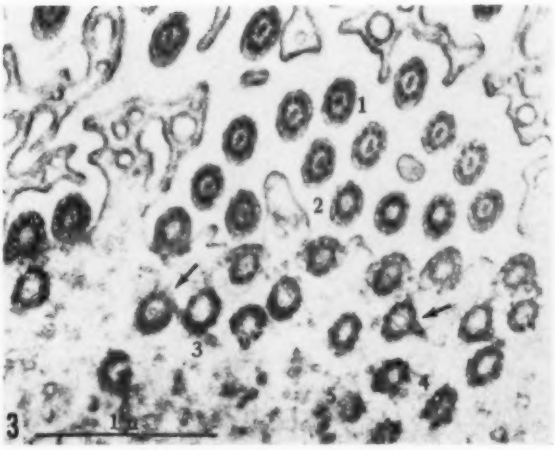


Fig. 3.—Cross section of cilia (top) and basal corpuscles (bottom) in the human tracheal epithelium. The free portion of each cilium which extends into the tracheal lumen is surrounded by the plasma membrane. Between the cilia are seen cross sectioned microvilli and protrusions of the cell surface, likewise bordered by the plasma membrane. Within the cell are basal corpuscles sectioned at different levels. The filamentous structures in the cilium and basal corpuscle can be traced as follows: 1) two central single ones and nine peripheral double ones; 2) just above the cell surface, the two central filaments disappear and the peripheral ones get closer together, acquiring a lateral contact with each other; 3) within the basal corpuscle, the lateral filaments become coarser, and in the central portion of the corpuscle is sometimes seen a circular profile (arrows); 4) the basal corpuscle tapers off and at 5) only a few filaments extend downwards into the cell body. Magnification 28000X.

cell is columnar and does not assume the goblet appearance. Contrary to the mucous granules, the serous granules take stain with osmium tetroxide like similar products of exocrine glands. Also, here, the Golgi complex of the cell is responsible for the production of the secretion granules which upon formation move toward the apical part of the cell. The top part of the cell starts to protrude and the serous granules are probably discharged one by one through a local temporary opening of the cell membrane like what is known to occur in the exocrine cells of the pancreas.

We have thus followed the formation of the mucous blanket and will now consider the means by which this blanket is kept moving toward the larynx. The main cell type of the tracheal epithelium is

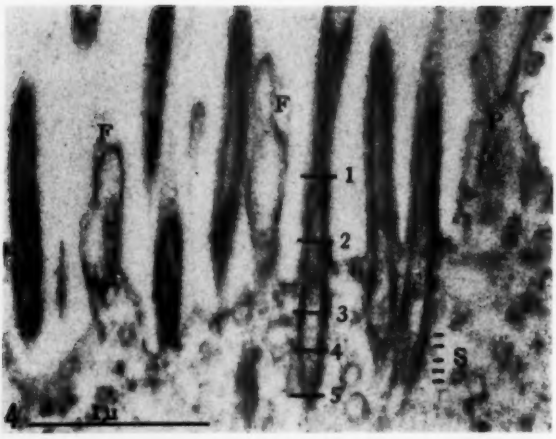


Fig. 4.—Longitudinal section of cilia and basal corpuscles in the human tracheal epithelium. Between the cilia are microvilli (F) and cellular protrusions (P). The numbers 1 to 5 correspond to the cross sections discussed in Figure 3. The rootlets of the basal corpuscle show cross striations (S) with a periodicity of about 625 A. Magnification 28000X.

the ciliated one. The cytoplasm is lighter than that of the mucous cells and the cell displays a nucleus, a small Golgi complex, and mitochondria, most of which are clustered beneath the cell surface with its abundant cilia.

The Golgi complex of the ciliated cell is mainly concerned with the protein synthesis within the cell itself. The mitochondria provide the energy necessary for this activity, but they certainly play a more important role in furnishing the energy required during the ciliary beat, which may explain why they are clustered so near the basal corpuscles of the cilia.

Each cilium emerges from its basal corpuscle into the tracheal lumen (Fig. 4). There are about 250-300 cilia on each cell and they are all covered by the thin cell membrane. The beat consists of the forward rapid stroke and the return slow stroke. The cilium is rigid during the forward stroke, whereas during the return stroke, it folds itself beneath the mucous blanket. The motor center for the ciliary beat is undoubtedly the basal corpuscle regulating the action of the cilium, be this a rigid rapid forward stroke or a folded slow return

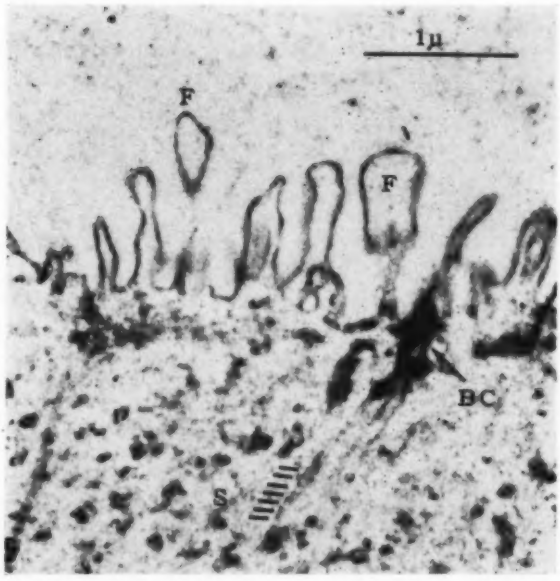


Fig. 5.—The luminal part of a brush cell in the human tracheal with an abundance of microvilli (F). Within the cell is a dense structure (BC) which has been interpreted as a basal corpuscle with attached tiny rootlets with cross striations (S). Magnification 23000X.

stroke. What do we find inside the basal corpuscle and the cilium to modulate this alternating action?

The basal corpuscle is composed of a central seedlike structure, not always present, and surrounding filaments which emerge into the free portion of the cilium as well as downwards into the cell body. The last mentioned fibers have been called rootlets and do not show any particular structure in the rat. In man, however, there is a marked difference as far as the structure of the rootlets is concerned (Fig. 4). These fibers have distinct cross striations distributed at regular intervals along their length with a periodicity of approximately 625 A. This structure thus reminds one of collagenous fibers which have the same periodicity, but in addition, a much higher complexity.

In the free portion of the cilium, the fibers are arranged longitudinally with two central and nine peripheral ones. They are all

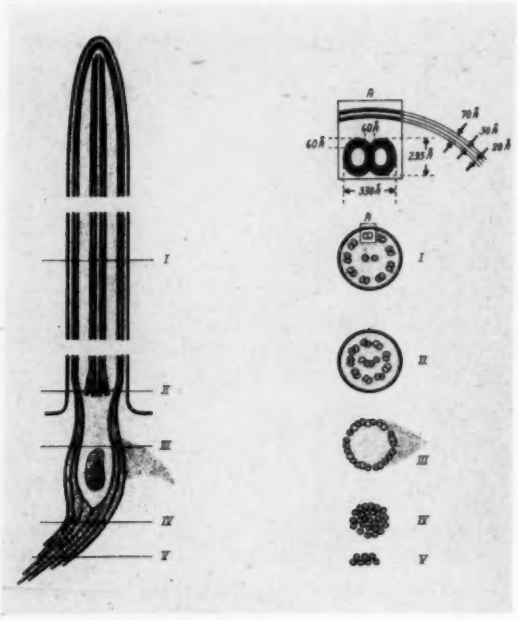


Fig. 6.—A schematic presentation of the ultrastructure of the tracheal cilia in the rat. For description see Figures 3 and 4. (After Rhodin & Dalhamn, 1956)

enclosed by the cell membrane represented by a thin dense single line. At the very top of the cilium, the peripheral filaments fuse and contact is probably also established with the central ones. In a tangential section of a local bulge of the ciliated cell top (Fig. 3), the cross sectioned free cilia and the basal corpuscles can be studied to advantage simultaneously and the pattern of the cross cut peripheral and central filaments can be analyzed at different levels in the same picture. It can now be appreciated that the number of the peripheral filaments is nine, a feature typical for cilia throughout the animal kingdom, and also found in the sperm tail. At a very high magnification, it will be found that each of the peripheral filaments actually is composed of two subfilaments in close apposition, whereas the central two filaments mostly stand out as single structures. The core of each filament is lighter than the dense wall which seems to form the structural unit of all filaments. In the basal corpuscle, the peripheral filaments split into several units, and as such get in a lateral contact with each other

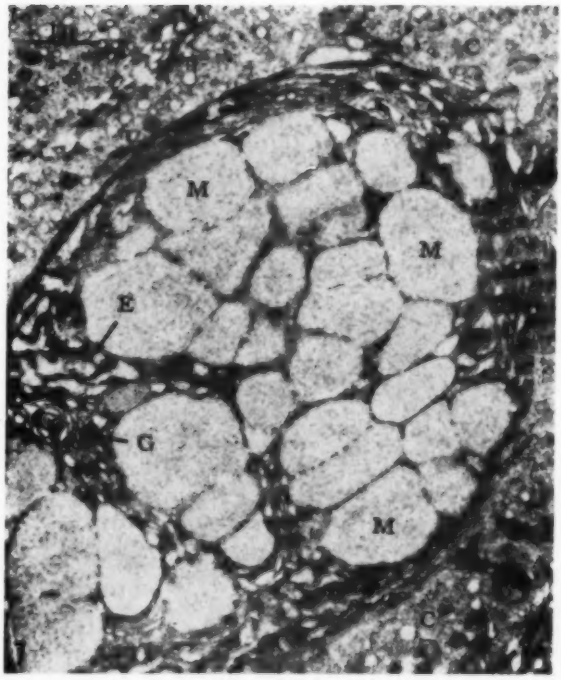


Fig. 7.—Cross section of a mucus cell (Goblet cell) in the human tracheal epithelium, surrounded by several ciliated cells (C). The mucous granules (M) are abundant sometimes coalescing, and take but little stain with osmium tetroxide because of their high content of mucopolysaccharides. The mucous granules are presumably formed by a co-ordinated action of the Golgi-membranes (G) and the α-cytomembranes (E) which also are referred to as endoplasmic reticulum. Magnification 11200X.

before they finally form the rootlets. The central two filaments split each into two subfilaments just before entering the basal corpuscle in which they end approximately above the central seedlike structure (Fig. 6).

Thus, it seems that the nature of the forward and return stroke can be explained by the complexity of structures discovered in the interior of the cilium and the basal corpuscle. Without going into any profound considerations about the function of the peripheral and central filaments, it is obvious that both the rigidity of the rapid forward stroke and the flexibility of the return slow stroke is ac-

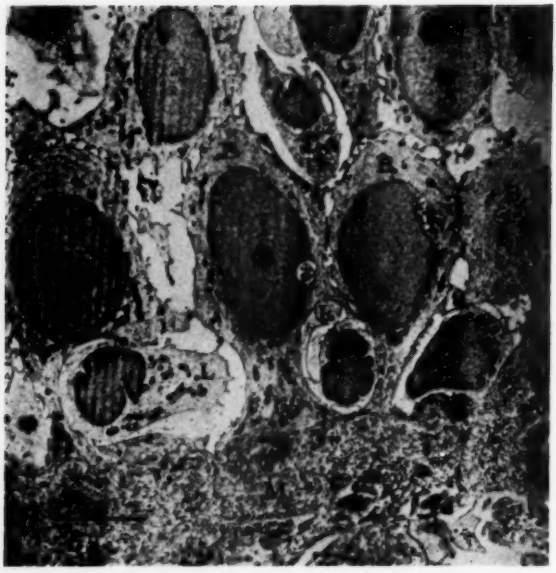

Fig. 8.—Basal cells (B) of the human tracheal epithelium resting on the basement membrane (M) which has an average thickness of seven microns. The basal parts of some ciliated cells (C) and one mucous cell (G) are seen indicating their inability to reach the basement membrane. In the spaces between the basal cells (B) are some lymphocytes (L). Magnification 3100X.

counted for when considering the system of fibers, forming a continuity from the basal corpuscle to the tip of the cilium, be the function of the fibers a contractile or an impulse-conducting one, or a mixture of both.

During the analysis of the rat tracheal epithelium, a peculiar cell was discovered, the features of which did not tally with any other known. It had a dense cytoplasm, small mitochondria scattered throughout the cell and numerous small extensions on the surface of the cell. The surface structures were easy to discriminate from regular cilia and also the microvilli of nearby mucous cells are of a different order of magnitude. At high magnification, the cell extensions of this particular type of cell stand out as either clumsy protrusions of the cell surface as single filiform projections. The function of the cell is rather obscure, but comparing the cell surface with known structures of other cell types, the extensions very much remind one of

the brush border extensions of the intestinal cells. Thus, judging from the similarity, one would expect that this particular cell would have resorptive functions and actively participate in regulating the viscosity of the mucous blanket.

Cross sectioned protrusions and single extensions at high magnifications are, however, puzzling, and one sometimes gets the impression that they represent the precursors of forming cilia. The cell would then be a newly formed ciliated cell.

Even more suggestive are pictures which are found in the human trachea, where a number of brush cells ordinarily can be identified, some of which have indications of a basal corpuscle with single root fibers (Fig. 5). Here, we might say that the brush cell is transformed into a ciliated cell, though it cannot be denied that the other brush cells without any sign of fragmentary basal corpuscles may have a resorptive function.

The source of cells with the potentiality of being converted into any kind of tracheal epithelial cells may be found close to the basement membrane where the so-called basal cells may be found (Fig. 8). In the rat, all cells of the epithelium rest on the basement membrane, whereas in man, only the local cells establish this contact.

In the rat, the basement membrane is a homogeneous, 800A thick, continuous structure. This is also true in man, but adjacent to its subepithelial surface is found a layer of densely packed collagen fibers with a random orientation. The close arrangement of collagen fibers extends approximately seven microns downwards where a more loose appearance prevails.

Our investigations thus have led us to a better understanding of the mechanisms involved in the formation of mucus, in its transport on the tracheal lining, and in the regulation of its viscosity. A schematic summary of the findings in the rat trachea is presented in Figure 1, its epithelium being of a simple columnar type as compared to the pseudostratified epithelium of the human trachea. It seems that we have now a rather extensive knowledge of the normal appearance in both rat and man which will represent the structural basis for extended studies on the pathology of the trachea and the bronchi. These studies may be performed preferably on biopsy material, as the electron microscopist is content if he gets only a small piece of tissue. And as the preparation technique for electron microscopy is not too complicated, it seems that in the near future we may have a useful

tool in the electron microscope in what would be considered the surgical electron pathology of the trachea and the bronchi.

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LXVIII

# THE COCHLEAR POTENTIALS AND THEIR RELATION TO HEARING

ERNEST GLEN WEVER, PH.D.
PRINCETON, N. J.

Since the discovery of the electrical potentials three decades ago there has been repeated inquiry as to their functions in the process of hearing. These potentials are of two types, those arising in the cochlea and those of the auditory nervous system at its various levels. This discussion is concerned with the first type, the cochlear potentials, and their particular relations to the auditory nerve responses and the process of sound perception.

Two distinct views have been expressed regarding the role of the cochlear potentials. One is that they are incidental, or a sort of epiphenomenon, and are not signficantly concerned in the hearing process. The other view, which is the one defended here, is that these potentials constitute one of the essential links in the chain of events from the entrance of sounds into the ear to the arousal of auditory sensations. The chief arguments may be stated briefly as follows:

1. The cochlear potentials are invariably present when the ear is functional. When they are absent, as has been observed in albinotic cats,[1] a strain of Dalmatian dogs,[2] waltzing guinea pigs,[3] and dancing mice,[4] the animals are also deaf by behavioral tests.

2. There is strong evidence that the cochlear potentials arise in the hair cells of the organ of Corti, which are the essential sensory receptor elements. When the ear is exposed to sounds of excessive magnitude the cochlear potentials suffer serious reductions from which there is but slight recovery, and sometimes they show a progressive deterioration over the course of several weeks. When these ears are studied histologically there is always a destruction or alteration of the hair cells. Other cells of the organ of Corti are damaged also when the exposure is extreme and prolonged, but when the exposure is more

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moderate the visible changes are restricted to the hair cells, and at the minimum level of damage show themselves merely as an alteration of staining properties.

3. When the sounds are loud, but are not excessive, the cochlear potentials reach large values of a millivolt or two, even though the point from which they are recorded, such as the round window membrane or a drill hole though the bony cochlear wall, is in the immediate vicinity of only a small number of sensory elements. It is obvious that at the place of generation of the potentials, in the hair cells themselves, the potential must have a very large value. Hence it can hardly fail to excite the nerve fibers that form a meshwork about the lower portions of these cells.

There are several experimental approaches to the problem of the functional role of these potentials. The one to be employed here is a comparison of the threshold acuity of animals as determined by behavioral methods with the pattern of cochlear potentials that may be recorded from the same animals. At this time only the potential pattern as recorded from the round window membrane will be considered. Perhaps at some future date the same comparison may be made with the patterns obtained from points in the interior of the cochlea, but at present there are not enough data for this purpose.

It is necessary to bear in mind the limitations of the cochlear potential patterns as obtained with an electrode on the round window membrane. This position is at the extreme basal end of the cochlea, and hence it reveals most prominently the activity of this region, and is not a true picture of the whole sensory process. The responses to the highest tones, which most actively involve the basal regions, are better represented than those to lower tones which more vigorously involve the apical regions. It happens, however, that the low tones always involve the basal regions in considerable degree, even though their main actions may be elsewhere. Indeed, every tone affects all parts of the basilar membrane and hence all of its hair cells to some extent. Accordingly this method of recording gives an indication of the actions of all tones, even though it is a biased indication.

In the following treatment the available data on auditory acuity and cochlear potentials have been brought together in all the animals

in which these two have been studied with sufficient exactness. Though the two functions have been considered in a great variety of animals from fishes to mammals it is only in a few that the quantitative requirements have been met satisfactorily. In some species like man, chimpanzees, and rats, there are good measures of acuity but little or nothing on the cochlear potentials. In others, such as frogs, turtles, and snakes, we have considerable data on the potentials but no satisfactory information on auditory acuity. In some other species there are partial data of both sorts, but not enough to permit a valid comparison. The only animals that satisfy the conditions sufficiently well to merit consideration at this time are marmosets, rhesus monkeys, cats, guinea pigs, and pigeons. This discussion is in part a plea for better behavioral and electrical measurements in other animals, and especially for greater uniformity and precision in experimental techniques. The most troublesome feature of many of the studies reported in the literature is a lack of care in the handling and measurement of sound stimuli. Many studies have lacked any sort of calibration of sound intensities, though a crude calibration is always possible by placing a group of normal human subjects in the sound field, and then expressing the results in terms of sound pressure as already determined for human ears. This is not the equal of an instrumental calibration, but it serves the purpose fairly well. Some of the acuity curves reproduced here were obtained with this procedure. More serious is the handling of the sound field, which often is irregular because of the presence of reflections from the walls of the cage or its surroundings. Then it is difficult to know what sound pressure is effective at any moment, for a small displacement of the head may alter its value.

*Marmosets.* First to be considered are results from a group of five marmosets whose acuity was tested behaviorally by Seiden[5] and whose cochlear potentials were later measured by Wever and Vernon.[6]

Conditioned responses to tones were produced by a shock-avoidance method. The animal was placed in a rectangular cage, and when it was at one end of this cage a tone was delivered and followed soon by an electric shock administered through the half of the floor on which the animal was standing. The shock could be avoided by the animal's moving to the opposite half of the cage, and all the animals soon learned to leap across the midline as soon as the tone appeared. Thresholds were obtained by reducing the sound intensity until the

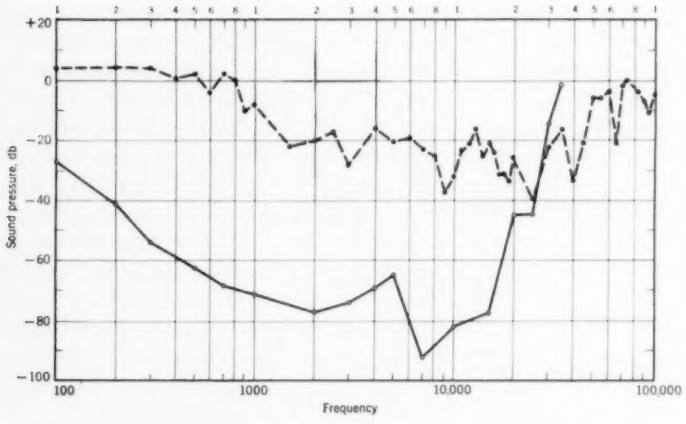


Fig. 1.—Auditory sensitivity in a marmoset as determined by behavioral and electrical methods. The ordinate shows the sound pressure in decibels relative to one dyne per sq cm at the threshold of hearing (solid line) and necessary to produce one microvolt of cochlear potential (broken line).

responses ceased. The results for one animal are shown in the lower curve of Figure 1. This curve indicates the sound pressure, in decibels relative to one dyne per sq cm, that was necessary to produce a response 50 per cent of the time.

The upper curve of this figure shows cochlear potential results obtained in one ear of the same animal. Here the curve represents the sound pressure required to produce a potential of one microvolt. The choice of level is somewhat arbitrary, and any other level would serve equally well provided that it be below the region of overloading of the ear. Because the cochlear potentials are a linear function of sound pressure except when overloading occurs, the shape of the curve does not change with the level. It must be emphasized that these potentials have no threshold, and are only limited at their lower range by conditions of observation. Under our conditions the measurements may be extended downward to about 0.05 microvolts, where they become obscured by physiological noise. There is nothing to be gained by working at the very low levels, and we have used a level of one

microvolt because it is high enough to give reliable results and yet does not require the presentation of excessively loud sounds.

Our interest is in a comparison of the shapes and limits of these two functions. The acuity curve falls progressively, representing an increase in sensitivity, from 100 to 2000 cycles per second. In this region the rate of increase in sensitivity is roughly proportional to the square of the frequency. Beyond 2000 cycles there is a rapid rise in the curve to 5000 cycles and then an abrupt fall to 6400 cycles, which is the maximum point of sensitivity, after which the curve rises rapidly to its upper limit at 35,000 cycles. The abrupt rise and fall in the region of 2000 to 6500 cycles is strikingly similar to the tonal dip found in certain human subjects (almost exclusively in males), and usually referred to as the "4096 dip." Every one of the five marmosets exhibited such a dip regardless of sex, with slight variations in the location along the frequency scale.

The cochlear potential curve is flat on the low-frequency end, and then declines at a fairly uniform rate of 500 to 25,000 cycles, representing an increase in sensitivity that is approximately proportional to frequency—not, be it noted, to frequency squared as is true for the acuity curve over much of this range. The greatest sensitivity in terms of these potentials is attained around 25,000 cycles, after which there is a fairly rapid decline, shown as a rise in the curve to the last point of measurement at 100,000 cycles. This limit of 100,000 cycles represents a practical limit of the apparatus used for producing and measuring the stimuli and for recording the potentials. It is possible that still higher tones would produce measurable potentials if these tones were available at sufficiently high intensities.

The cochlear potential curve contains some irregularities, especially in the high frequencies, but there is nothing corresponding to the tonal dip at 5000 cycles.

Particularly striking is the difference in the forms of the curves at their upper ends. The acuity curve rises almost abruptly from 7000 cycles to its limit at 35,000 cycles, whereas in this same region the cochlear potential curve is fairly uniform.

Thus there are five main differences between the behavioral and electrical functions: 1) the difference in slope at the low-frequency

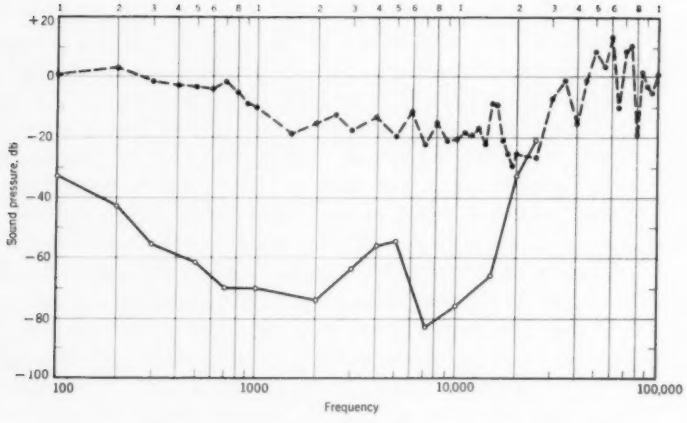


Fig. 2.—A comparison of acuity data and cochlear potentials for a group of five marmosets. The arrangement is the same as in the preceding figure. The same animals are represented by the two functions.

end, 2) the absence of a tonal dip in the cochlear potential curve, 3) the difference in the location of the maximum of sensitivity, 4) the difference in slope at the high-frequency end, and 5) the relatively early termination of the acuity curve at 35,000 cycles.

These differences were observed in much the same form in the remaining four animals also, as may be judged from Figure 2 which represents the mean data for all five. Here the uppermost point on the acuity curve is 25,000 cycles, because this was the highest frequency reached by all five animals. Actually the mean upper limit was 33,000 cycles; one animal stopped at 28,000 cycles and one went as far as 37,000 cycles.

*Rhesus Monkeys.* Results for the rhesus monkey are presented in Figure 3. The two lowermost curves represent acuity as determined by behavioral methods. Wendt[7] in 1934 trained monkeys of four different species to take food from a drawer when a tone was sounded, and the thresholds of the one rhesus (*Macaca mulatta*) of the group are shown by the long-dashed curve. This animal showed an upper

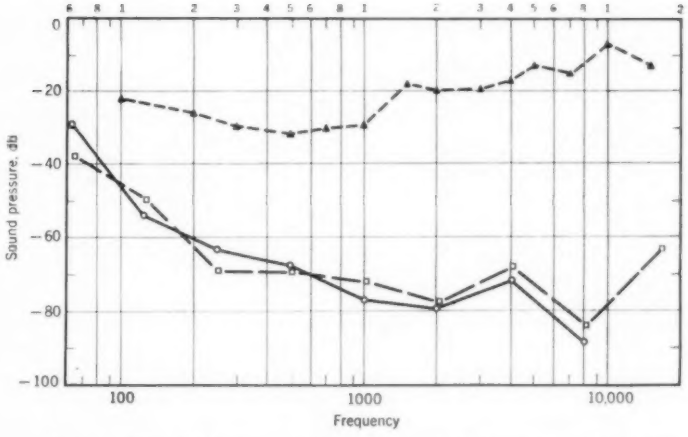


Fig. 3.—Threshold curves (solid and long-dashed curves) and cochlear potential data (short-dashed curve) for rhesus monkeys. These curves are for different animals.

limit of hearing of 33,600 cycles. Harris[8] in 1943 used eight monkeys of the *Macaca* genus, including two *Macaca mulatta* and three named as *Macaca rhesus*, and conditioned them to produce bodily movements in response to tones by delivering electric shocks when the tone was sounded. The mean thresholds of the group of eight are shown by the solid curve. There is good agreement between these two sets of data. The curves fall from 64 to 8000 cycles, except for a moderate rise at 4000 cycles, indicating a progressive improvement in sensitivity. The single point at 16,384 cycles indicates a considerable decline in sensitivity in this region.

The uppermost curve represents cochlear potential data obtained by Wever, Vernon, and Lawrence[9] in a group of three *Macaca mulatta* monkeys. This curve shows a slight decline from 100 to 1000 cycles and then a rise in the higher frequencies. The form is obviously different from the acuity function, and this curve is rising when the acuity curve is passing through its optimum region. Neither type of function has been carried far enough into the upper frequencies to determine a limit.

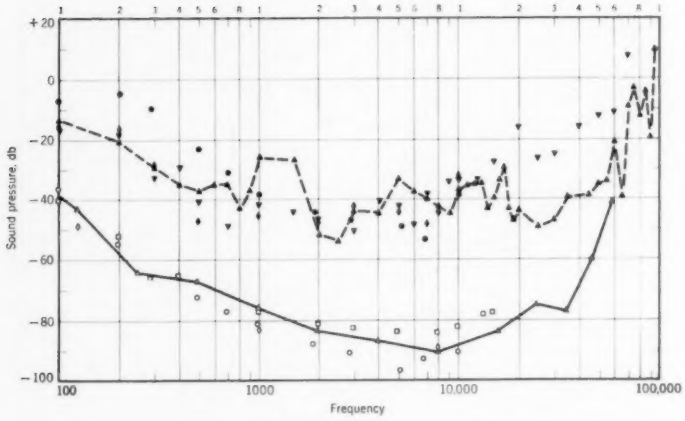


Fig. 4.—Thresholds and cochlear potentials for cats. The acuity data are from four sources (unfilled forms) and the cochlear potential data from three sources (filled forms).

*Cats.* Numerous observations by both behavioral and electrophysiological methods have been made on cats, and some of these are shown in Figure 4. Acuity data are represented from Dworkin et al[10] on several cats (open squares), from Kryter and Ades[11] on a group of 20 (open diamonds), from Neff and Hind[12] on three (open triangles), and from McGill[13] on three (open circles). There is fairly good agreement among these data, with McGill's animals showing somewhat greater acuity above 500 cycles. The curve declines at a rate that is nearly proportional to the square of the frequency over the range from 100 to 1000 cycles, and then more slowly to its maximum of sensitivity around 8000 cycles. Thereafter it rises, at first slowly and then more rapidly, to a limit at 60,000 cycles.

The cochlear potential data represent observations on the same three animals used by McGill in his behavioral study (solid circles), together with measurements on one cat by Wever and Bray[14] in 1938 (solid diamonds) and on two others by Wever, Vernon, Rahm, and Strother[15] in 1958 (solid upright and inverted triangles). The dashed curve is drawn through the solid upright triangles, and represents one

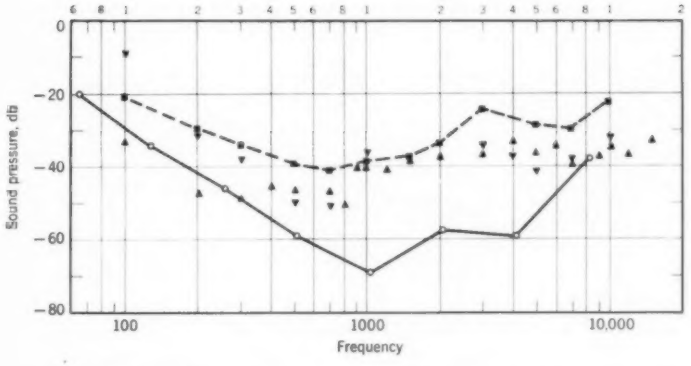


Fig. 5.—Thresholds and cochlear potentials for guinea pigs. The open circles represent acuity data for a group of 7 animals, and the closed forms represent cochlear potential data from three sources.

of the animals for which observations were made in the very high frequencies. These data show some variations, but it is clear that there is a progressive gain in sensitivity from 100 to 2000 cycles, at a rate nearly equal to that shown behaviorally, and then a fairly level region to 45,000 cycles after which there is a rapid loss. Most cats tested in the high frequencies gave responses up to 100,000 cycles. The maximum of sensitivity is not well defined, but probably lies in the region from 2000 to 7000 cycles.

The two functions are fairly similar in form in the low frequencies, but differ in the region of maximum sensitivity and in the upper limits attained.

*Guinea Pigs.* The solid curve of Figure 5 represents the mean auditory thresholds of seven guinea pigs studied by Horton[16] by a conditioned response method. The curve declines from 64 to 1024 cycles at a rate proportional to the square of the frequency, attains its maximum of sensitivity at 1024 cycles, and then rises. No measurements were made beyond 8192 cycles, but it is certain that guinea pigs hear much higher tones.

The cochlear potential data are from three sources. The filled inverted triangles represent measurements on one ear by Wever and

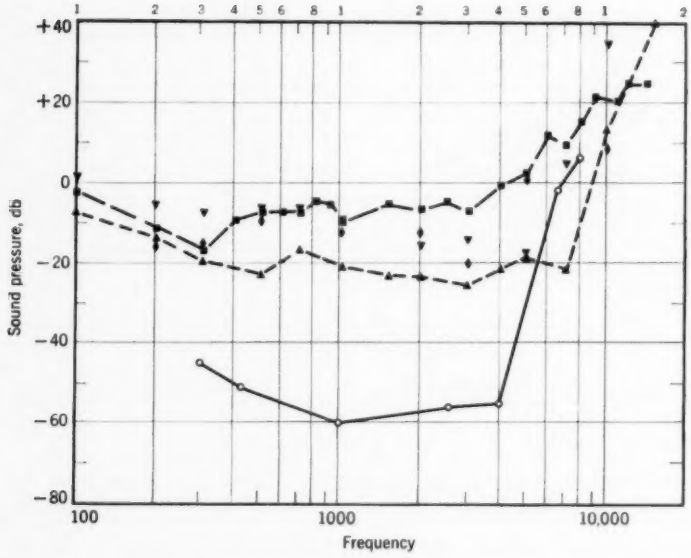


Fig. 6.—Thresholds and cochlear potentials for pigeons. The open circles represent acuity data for a group of five animals, and the closed forms represent cochlear potential data from four sources.

Bray[17] in 1936, the filled upright triangles the means of four ears studied by Wever, Lawrence, and Vernon (unpublished), and the squares the means of 35 ears studied by Lawrence and Yantis[18] in 1957. The cochlear potential function seems to reach its maximum of sensitivity in the region of 500 to 800 cycles, and then rises thereafter. Potentials have been observed in guinea pigs to tones as high as 35,000 cycles, but no detailed study has been made in the uppermost frequencies.

*Pigeons.* The solid curve of Figure 6 shows mean thresholds for a group of five pigeons studied by Heise[19] in 1953. The birds were trained to peck at a key, and were rewarded if this was done in the presence of a sound. After a long period of practice they pecked more rapidly when the sound was heard. The sensitivity improves from 300 to 1000 cycles, remains nearly constant to 4000 cycles, and

then rapidly falls off to the limit at 8000 cycles. An early study by Wassiljew[20] indicated an upper limit between 10,427 and 12,401 cycles.

The cochlear potential data represent one ear studied by Wever and Bray[21] in 1936 (diamonds), another studied by Bray and Thurlow[22] in 1942 (inverted triangles), the mean of 12 studied by Wever, Vernon, and Lawrence (squares), and the mean of eight studied by Wever and Vernon (upright triangles). Curves are drawn through the last two sets of data (not previously published). In general, there are only slight variations of sensitivity from 100 to 4000 cycles, and then a considerable decline in the uppermost frequencies.

The acuity and cochlear potential curves are not very different in the middle range, but the acuity curve rises more rapidly in the high frequencies and reaches a limit at a point where the potentials have not suffered much loss.

## COMMENT

These results present a number of features that bring out certain systematic relations between the cochlear potentials and auditory acuity.

1. *The Rise in Low-Tone Sensitivity.* In general, the sensitivity as determined behaviorally improves rapidly in the low-frequency region, at a rate that usually is proportional to the square of the frequency. It is pertinent to mention that in man this rate is significantly more rapid, being proportional to the cube of the frequency. This disparity between man and the other animals needs further investigation.

The sensitivity as shown by the cochlear potentials does not increase as rapidly over the lower frequency range as the threshold acuity; in general it improves about in proportion to the frequency. Here the cat is exceptional in that the improvement is more rapid.

The greater rate of rise of threshold acuity as compared with the potential function no doubt reflects mechanical conditions in the cochlea and activities of the auditory nervous system that serve to enhance the action of the high tones. These conditions, according to

an earlier analysis,[23] are four in number: the progressive narrowing of the cochlear response pattern, the varying density of innervation along the cochlea, the changing excitability of the nerve fibers to pulsating currents (*i.e.*, to the cochlear potentials), and the rising rate of nerve impulses as a function of frequency. The narrowing of the response pattern for the high tones represents a concentration of the available sound energy upon a limited number of sensory cells. This is an aid to acuity because the thresholds of these cells are more readily crossed than if (as happens at lower frequencies) the same energy were distributed more uniformly along the cochlea. It has no corresponding effect upon the cochlear potentials because at low levels these potentials reflect the total sound pressure without much relation to its spatial distribution.

2. *The Region of Maximum Sensitivity.* The acuity functions usually show a fairly well-defined region of maximum sensitivity. For the marmoset this is at 7000 cycles, for the monkey and cat at 8000 cycles, and for the guinea pig and pigeon at 1000 cycles. However, for the pigeon the curve is rather flat from 1000 to 4000 cycles, and the data on the monkey are somewhat scanty, especially at the high-frequency end.

The cochlear potential curves are generally much flatter, and it is difficult to designate a maximum. In the marmoset there is a broad region from 1500 to 25,000 cycles where the sensitivity is high, with the best point around 25,000 cycles. In the monkey this region is from 300 to 1000 cycles, in the cat from 1000 to 40,000 cycles, in the guinea pig from 500 to 3000 cycles.

It is clear that the two types of function do not agree in the locations of their maximums. In all but the monkey the acuity maximum is reached and then the curve rises rapidly in a region where the cochlear potentials are either growing more sensitive or at least are maintaining their sensitivity.

3. *The Upper Limit.* Just beyond the maximum the acuity functions rise rapidly and finally reach a limit beyond which no responses could be observed at the sound intensities available. The limits attained were 33,000 cycles for marmosets, 33,600 cycles for a monkey, 60,000 cycles for cats, and 8000 to 12,000 cycles for

pigeons. No upper limit has been determined for guinea pigs, and the monkey data are limited.

The cochlear potentials extend much beyond the acuity curves in the marmoset and cat, the only mammals in which the high-frequency responses have been explored thoroughly.

In the pigeon the two limits are not very different. The highest frequency for which cochlear potentials have been obtained is 15,000 cycles, and this observation requires large sound pressures of 100 dynes per sq cm.

From these results it appears that the ear is limited in the extreme high-frequency range by both mechanical and neural conditions. A basic condition is the increasing mechanical impedance of the conductive mechanism and especially that part within the cochlea itself. This condition shows itself in both the cochlear potential function and the acuity function.

Other conditions apply more seriously to the acuity function. The factors that operate so as to enhance the threshold sensitivity as frequency rises evidently reach a limit. Primary in this regard are the progressive narrowing of the response pattern and its shift basalward as the frequency rises. The basal shift finally brings the main response area into a region of the cochlea where the innervation is sparse. The excitability of nerve fibers by pulsating currents probably does not continue to rise with frequency, but attains a limit, and the rate of nerve impulses reaches a limit also. For all these reasons the acuity function fails while the cochlear potential function is still continuing.

110 Eno Hall

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LXIX

# VOCAL REHABILITATION BY AUTOGENOUS VEIN GRAFT

JOHN J. CONLEY, M.D.

NEW YORK, N. Y.

When we realize that Professor Theodor Billroth of Vienna performed the first successful total extirpation of the larynx in 1873, and that shortly thereafter an artificial larynx of the vibrating-reed variety was offered to the laryngectomee, we must admit that very few fundamental advances have been proposed in the past eighty-six years to facilitate the patient's vocal rehabilitation. More than 2,000 laryngectomies are performed each year in the United States, which indicates general acceptance of the technique. Yet the threat of the loss of the voice in some instances has created such anxiety, that the patient may delay his operation or seek a less effective type of treatment.

The greatest advance in re-orienting the laryngectomized patient has been the development of the esophageal voice. Forty per cent of the patients, however, discontinue training against the advice of their therapists. The discipline, practice and effort required to speak again may be rewarded with a new voice of completely adequate quality. Most individuals, however, fall short of this goal.

Only 7% of the laryngectomees have accepted the assistance of any type of mechancial device. I am certain, however, that if the technical aspect of these instruments of artificial phonation could be improved, patients would be anxious to adopt them.

In 1958 a new surgical technique for the vocal rehabilitation of the laryngectomized patient was presented.[1] It consists of the creation of a mucosal tunnel, fashioned from the anterior wall of the cervical esophagus and positioned in such a way as to fulfill the criteria which were determined to be essential for the success of the operation. A review of 15 cases was given. This new report is a modification and development of that concept.

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From the Surgical Service of St. Vincent's Hospital, and the Head and Neck Department, Pack Medical Group, New York City.

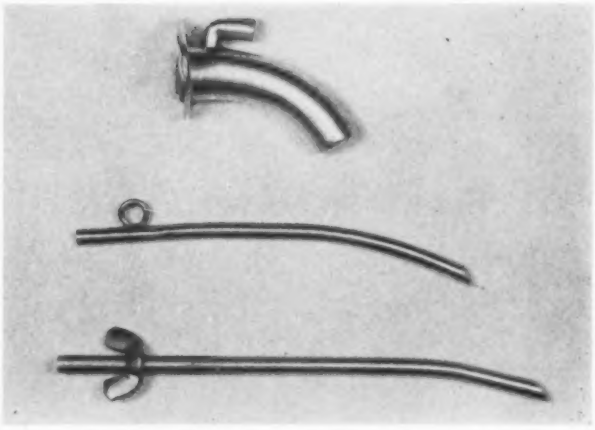

Fig. 1.—Special laryngectomy tube. Trocars through which vein graft is inserted.

The indications for the operation include patients undergoing laryngectomy or laryngectomy and neck dissection for the treatment of cancer, as a primary procedure. It may also be applicable as a secondary elective procedure on individuals who have undergone these extirpative techniques, and who have never been able to master esophageal speech and who reject the artificial larynx, and yet persist in their desire to speak. The new technique carries an increased risk of failure in individuals with a devitalized tissue bed secondary to irradiation.

The fundamental problem was to create a patent communication which would permit the free flow of air from the trachea into the gullet without creating the fistula phenomenon. In this particular instance, it is accomplished by the implantation of an autogenous vein 8 cm in length, connecting the skin of the neck just above the tracheal stoma with the lower portion of the cervical esophagus through a passageway in the tracheo-esophageal fascia. The great saphenous vein has proved satisfactory for grafting purposes. When the vein graft is implanted at the time of the primary operation, the technique is greatly simplified by threading a No. 8 catheter or plastic tube through its lumen, and then inserting this into a specially adapted trocar which is threaded through the fascia between the trachea and esophagus. After penetrating inferiorly for approximately 6 cm, the

Fig. 2.—Special laryngectomy tubes.

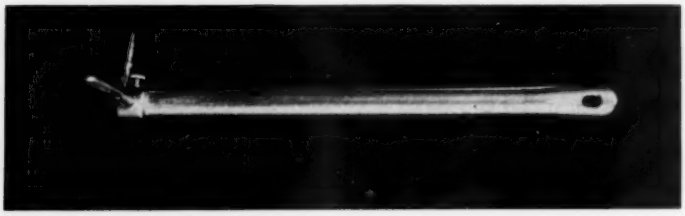

Fig. 3.—Perforated short esophagoscope.

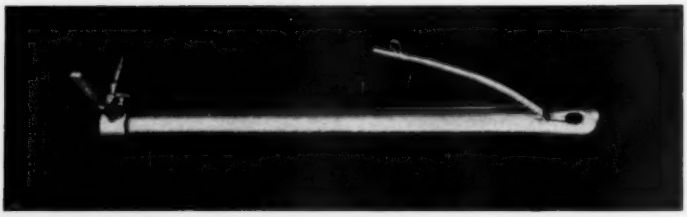

Fig. 4.—Trocar in position to perforate into esophagus.

anterior wall of the cervical esophagus is punctured with the special trocar. The trocar is then removed leaving the vein graft in position, with the catheter remaining in its lumen. It is fixed to the anterior wall of the cervical esophagus with four atraumatic No. 3-0 chromic catgut sutures. Its external opening is then adapted to the skin just above the tracheal stoma. Ordinarily it does not communicate with the repair of the pharyngeal mucosa and does not complicate the

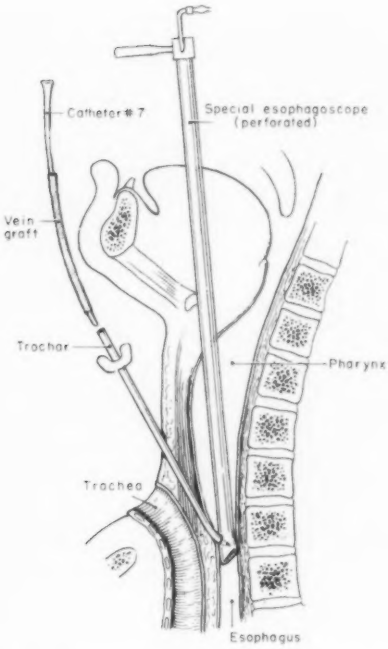


Fig. 5.—Special esophagoscope in position for cannulization with trocar and then vein graft.

excisional phase of the operation. A special tracheostomy tube with a vent completes the connection.

When this technique is carried out as a secondary and separate procedure, the vein is threaded through the same fascial space in precisely the same manner and the perforation is made through a fenestrated short esophageal speculum, which is in the esophagus as a guiding instrument.

This technique was carried out on 15 patients. By its very nature, it is quicker and easier to accomplish than the creation of a mucosal tunnel. Two individuals had from slight to moderate fistula activity with leak of saliva and occasionally of food. Five individuals had spontaneous closure of the vein graft. They were regrafted and one closed a second time. This tendency towards spontaneous atresia of the tunnel is unquestioned and distressing. It is the natural conse-

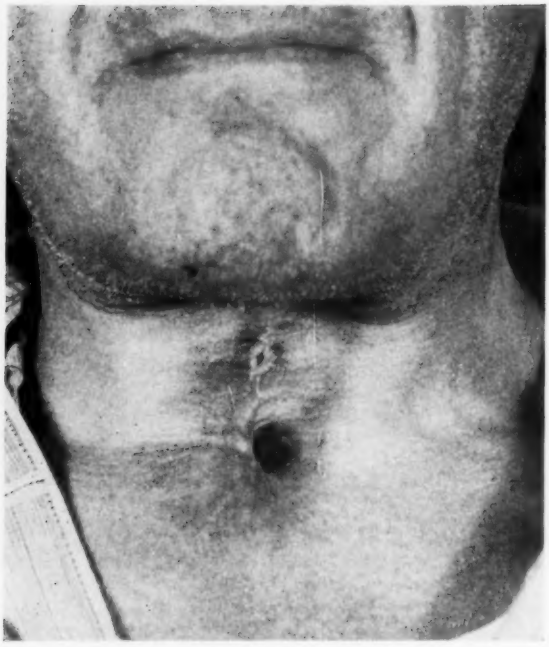

Fig. 6.—Air tunnel opening on neck.

quence of irritation and low-grade infection of the intima of the vein graft when the endothelial surfaces are coapted. In order to overcome this, a small catheter or plastic tube must be maintained in the lumen of the vein graft to preserve patency. This responsibility must be assumed by the patient during the intervals away from the office. This has proven to be a disadvantage. If the patient, as has occurred in some instances, has permitted the catheter to remain out of the vein graft for an interval of two days, atresia will have occurred and recannulization as an office procedure has not been successful.

Speech may be attempted as soon as the primary wound is healed. Special instructions are not necessary as the patient shunts air from the trachea into the gullet by placing his finger over the special laryngectomy tube. Some ingenious adaptations of this maneuver have been carried out by various patients with ball valves, elongated plastic tubes, etc.

Quality of the voice is essentially that of pharyngeal or esophageal speech. There is, however, a greater supply of continuous air, greater ease of the production of the sound, better air and voice control, and an absence of the gulping phenomenon.

It has assisted all patients in understanding esophageal speech technique. When esophageal speech has been attained by self-training, the patient may use the two methods in combination, or permit the graft to close spontaneously.

Speech analysis by the Bell Telephone Laboratories through recordings, word scores, sentence intelligibility, sound techniques and spectrograms, indicated that approximately 98 per cent of the sentences spoken by the patient tested could be understood. This is a high rating for the postlaryngectomized patient and compares favorably with any esophageal speakers, and, indeed, with some individuals who have a normal larynx. The report also included the fact that "these patients put together longer groups of words before pausing for more air, and in this respect approach a natural speaker in forming his sentences."

## CONCLUSIONS

1. A technique of autogenous vein graft has been presented for vocal improvement in the laryngectomized patient.

2. In essence, the vein graft permits air to pass by special apparatus from the trachea into the gullet without the fistula phenomenon.

3. The procedure is simpler than the mucosal tunnel technique. There is a significant tendency for the vein graft to become atretic.

4. Patients' emotional reaction to the basic concept of this operation is favorable.

5. Further investigation in speech rehabilitation and technical aids for the laryngectomee is warranted.

139 East 36th Street

Special thanks is extended to Doctor M. H. Lurie for his suggestions and help in carrying out this investigation.

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LXX

# EXPERIENCES WITH THE FENESTRA OVALIS TECHNIQUE OF SHEA

## A PRELIMINARY CLINICAL AND EXPERIMENTAL REPORT

DAVID MYERS, M.D. S. D. ERULKAR, D.PHIL.
WOODROW D. SCHLOSSER, M.D. LINDSAY PRATT, M.D.
RICHARD A. WINCHESTER, PH.D.
PHILADELPHIA, PA.

During the preceding year, the Fenestra Ovalis Technique of Shea[1,2] has been used in this department to an increasing extent for the relief of deafness due to otosclerosis. Sufficient clinical and experimental data have now been accumulated to make it possible to present this discussion of the value and potential of this most recent development in otosclerosis surgery, as well as to permit the senior author to present a series of rather detailed observations regarding the surgical procedure itself.

Elsewhere, Myers, Schlosser and Winchester[3] have described the results of five years' experience with stapes mobilization surgery, and have presented some conclusions concerning the surgical management of the otosclerotic patient. Since the preparation of this report, however, additional experience and information have been obtained which form the basis for the present paper.

The opportunity to do a substantial number of revisions of former stapes mobilization surgery cases has afforded an excellent means of evaluating the results of this surgery, and to engage in further clinical observation of the otosclerotic disease process itself. Many patients who previously appeared to have a normal, blue footplate were later found on revision when the footplates were removed, to possess a far more advanced degree of otosclerosis than was originally suspected. Furthermore, biopsies of otosclerotic ossicles have been obtained in a majority of these cases. Pathological studies have shown that every footplate, and almost every crus studied showed definite evidence of the presence of otosclerotic invasion (Fig. 1).

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From the Department of Otorhinology, Temple University Medical Center.

In addition, the removal of the footplate makes it possible to ascertain the extent of the otosclerotic lesion in the fossa ovalis more readily. These observations have led to the conclusion that approximately 60 per cent of the patients receiving otosclerosis surgery present the diffuse type of otosclerotic formation which, experience has shown,[3] requires surgical intervention greater than that afforded by the simpler, more conservative stapes mobilization techniques employed in the past. It would seem then, that otosclerosis surgery is now at a major crossroads with respect to the means whereby the patient with diffuse otosclerosis can be given a significant and lasting improvement in hearing acuity. Should the operation of choice be the lateral canal fenestration, which has been tried and proved of great value over the last two decades, or is the answer to be found in the newer fenestration of the oval window procedure? The problem does not admit of easy solution, but it is hoped that the present discussion will be of value with respect to the formulating of a final answer.

It would seem that two important factors are involved in any decision regarding a choice of method of re-establishing sound transmission into the inner ear of the otosclerotic patient: 1) The nature of the surgical technique and the patient's reactions to the procedure and 2) the comparative effectiveness of the technique in question in producing a) hearing acuity at the socially practical level and b) significant amounts of improvement in acuity in those patients whose hearing by bone conduction precludes reaching the 30 db hearing level, that is, the Class D surgical candidate.

Since the addition of the fenestra ovalis technique to our surgical armamentarium, the following policy has been routinely followed: For the patient showing a localized otosclerotic lesion (Bellucci I, II), the by-pass technique is carried out. If, on the other hand, it is discovered at the time of surgery that the patient possesses diffuse and well advanced lesions which have covered and invaded the fossa ovalis (Bellucci III, IV), or if the patient presents an active otosclerotic disease process with fulminant degeneration and signs of cochlear involvement, or if evidence of extensive crural damage sustained in earlier surgery and separation of the incudo-stapedial joint or both of these are seen, the Shea fenestra ovalis procedure is done.

Preparatory to doing either the by-pass or the fenestra ovalis operation, the following procedures are carried out. These techniques are performed under local anesthesia and the patient is heavily sedated prior to coming to the operating room. The usual pre-operative rou-

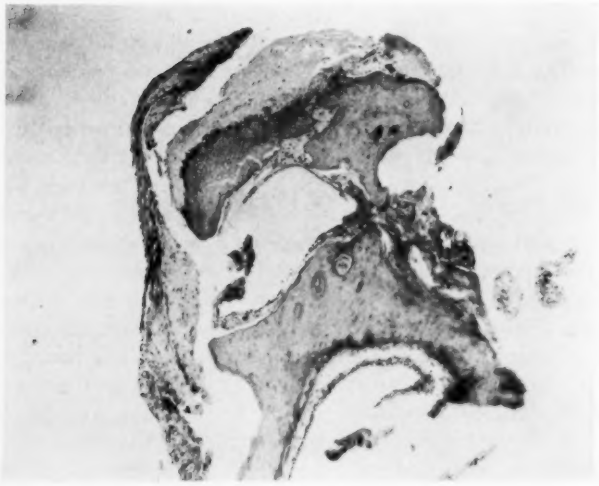

Fig. 1—Photomicrograph of a section through an otosclerotic stapes footplate showing bony tissue with thickened trabeculations and sclerotic change, X 65.

tine is as follows: 100 mgm of Nembutal® is given the night before surgery. On the morning of the operation, the patient receives an additional 100 mgm of Nembutal two hours pre-operatively, and 10 mgm of Compazine® one hour before surgery. Thirty minutes before the operation 50 to 75 mgm of Demoral® is given, and if the patient is not adequately sedated when reaching the operating room, an additional 30 mgm of Nisentil® is injected intramuscularly.

Upon entry into the operating room, the patient is properly positioned and draped on the operating table. The ear is prepared by filling the external auditory canal with 70% alcohol which is allowed to remain from 10 to 15 minutes. The patient's hair is held back with ointment or K-Y jelly in order to keep stray hairs from entering the operating field. In addition, the entire auricular region is washed with soap, ether and alcohol.

In order to obtain local anesthesia, the entire circumference of the meatus is injected with 1% xylocaine solution. This enables the surgeon to insert a much larger speculum into the canal without producing patient discomfort. With the speculum in place, the skin

of the auditory meatus adjacent to the drum is injected with 1% xylocaine solution containing 1:100,000 epinephrine. Because several severe epinephrine reactions have resulted in the past, the two types of solutions are used, with the second containing this very small proportion of epinephrine.

Then using the 10 power magnification of the operating microscope, the triangular incision of Shea is made in the posterior auditory meatal wall. It is necessary to continue this incision anteriorly and superiorly as far as the short process of the malleus, and inferiorly to the midportion of the canal so that the thinned skin flap can be folded forward and made to lie on the tympanic membrane, thus providing a wide exposure of the posterior region of the tympanic cavity. With this done, extensive dissection of the outer aditus wall is carried out using sharp curettes or surgical cutting burrs. This usually makes the stapes footplate clearly visible and accessible to surgical manipulation.

Once these procedures have been accomplished, the ossicular chain and area surrounding the fossa ovalis are inspected under 16 power magnification. With increasing experience, the extent and distribution of the otosclerotic foci are readily determined. Whenever diffuse or well advanced lesions are found, preparations are made to fenestrate the footplate of the stapes.

First, a tenotomy is done which further increases the visibility and accessibility of the footplate. Next, separation of the incus and stapes is carried out using a Shea pick to sever the capsule surrounding and binding together the lenticular process of the incus and the head of the stapes. Care should be exercised to avoid injury to the knob of the lenticular process, for damage to this structure increases the difficulties of securing a firm articulation between the incus and the plastic columella. In the event that damage is or has been done to this structure, a plastic columella can still be fitted by using a plastic tube of increased diameter (190 caliber) and shaping or notching its upper end to conform to the shape of the incus.

In order to remove the crura of the stapes, firm pressure is made in a downward and medial direction upon the stapes head using a Shea pick. This maneuver produces a fracture of the crura of the stapes which usually occurs flush with the footplate.

The next step involves the removal of the mucosa from the entire promontory, and from the horizontal portion of the facial nerve as

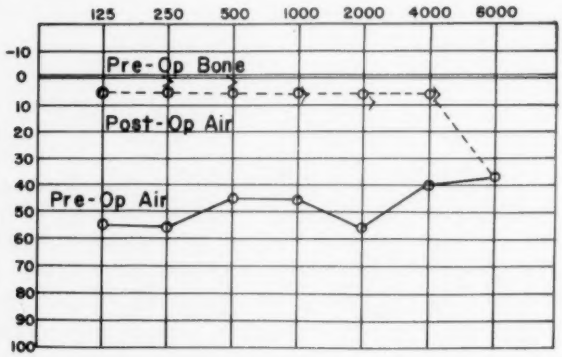


Fig. 2.—Audiometric results following a fenestra ovalis operation in a class A candidate.

far anteriorly as the area of the geniculate ganglion. The mucosa on the promontory is also removed inferiorly to the upper border of the round window. It is important to note that the mucosa covering the fossa ovalis itself is allowed to remain intact. This serves as an aid to the later removal of the footplate. In order to secure a dry operative field, Gelfoam sponges soaked in 1:1000 epinephrine in solution are placed upon the denuded promontory and allowed to remain there until the preparation and placement of the vein graft. In addition, all bleeding areas of the skin flap are treated with electrodessication or coagulation, for it is extremely important that blood does not seep into the operative site during the course of the fenestration of the oval window.

While these preparations are being carried out, an assistant removes a section of vein from the patient's forearm, taking care to obtain a segment of large enough diameter to cover, when split, the fenestra ovalis and the vein graft bed perviously prepared. Under the microscope the vein section is stripped of its attached areolar and adipose tissue and then split so that its intimal surface is exposed. When this is done the surgeon can determine if the vein graft is the proper size, or requires trimming to make it fit properly. The measurement can be made more exact by the use of the fenestrometer to indicate the exact size of the vein to be cut.

Once the vein graft is prepared, the gelatin packs are removed and the skin graft is placed in the hypotympanum so that it can be slid over the fenestra ovalis immediately after the removal of the stapes footplate. All is now in readiness for the fenestration of the footplate of the stapes.

If the footplate is seen to be very thick because of the invasion of the otosclerotic bone, a small Wullstein cutting burr on a special contra-angle attached to a Chayes hand-piece is used to thin out the footplate until a fenestration occurs. This can be seen taking place as a gray line appears which is similar to that developing during the fenestration of the lateral canal. As the drilling completes the removal of the otosclerotic footplate, the bone crumbles away revealing the patent fenestra. In very advanced cases of otosclerosis, overhanging projections as well as mounds of bony foci are encountered which cover and obliterate the fossa ovalis. These formations can also be removed using various sized diamond and cutting burrs. As a rule, the large otosclerotic masses anteriorly and inferiorly are removed, after which the footplate can be attacked. In order to prepare an efficient bed for the vein graft, it is necessary to saucerize the promontory and fossa ovalis so that a shallow cup-like excavation is produced.

At times the footplate is sufficiently thinned with the burrs to permit the use of a Shea pick to perforate and pick away the footplate until a large fenestra is obtained. Then, the opening in the footplate can be enlarged by inserting the 4-0 burr into the fossa ovalis itself. The drill is allowed to run very slowly, and should there be any bone dust deposited into the vestibule, enough perilymph fluid is present to create a paste of the dust which can be aspirated. Whenever possible, the fenestra ovalis which is produced should follow the outlines of the fossa ovalis. Fine Shambaugh picks are useful in removing any small bony projections or spicules which remain on the rim.

Earlier, when we attempted to remove the footplate as a whole, two were lost in the vestibule. As a result, labyrinthitis and subsequent tissue organization took place which caused a loss of cochlear function. Therefore, it is advisable to remove the footplate piecemeal and to make certain that all remaining bone fragments are removed from the fossa ovalis. The residual mucosal covering left by the operator provides a very useful means of grasping and removing these fragments.

Of necessity, the fenestra ovalis technique produces a small amount of trauma to the inner ear. Strict control of surgical technique, however, can minimize this reaction to a considerable degree. In particular, suction in and around the fossa ovalis should be avoided as much as possible so as to remove a minimal amount of perilymph.

Several comments should be made regarding the placement of the vein graft. First of all, the vein graft should be positioned over the

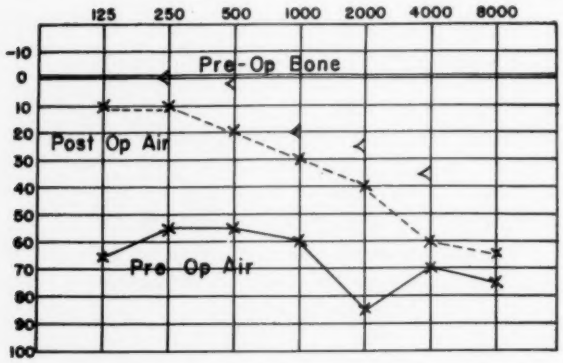


Fig. 3.—Audiometric results following a fenestra ovalis operation in a class B candidate.

fossa ovalis immediately after the window has been opened and prepared. This insures that there is little chance for further irritation of the inner ear structures to occur. In addition, the depth of the fossa ovalis should determine the thickness of the graft. A very deep fossa requires a thin graft, while a thicker section is needed in the case of a shallow fossa. Finally, it should be stressed that the vein graft must cover a large area surrounding the fossa ovalis so as to insure that it will be fixed to the bone in order to avoid undue shifting during the positioning of the plastic prosthesis.

Some degree of skill is required in the preparation of the polyethylene plastic tubing. Usually, a 90 caliber diameter provides the correct degree of rigidity for the columella. The tubing can be cut to size at the time of surgery, or obtained in several pre-cut lengths from the Richards Surgical Company of Memphis, Tennessee. These lengths are 3, 3.5 and 4 mm. A special measuring probe has been devised by the Richards Company at the request of the senior author which makes it possible to measure the size of the tube section more accurately. Once it is determined that the tubing is of the proper length, a small bevel should be cut on one end. The beveled end fits into the oval window and faces the lower border of the fossa ovalis. Again, the diameter of the 90 caliber tubing in most instances provides a very secure fit between the tube end and the lenticular projection. This condition must be obtained to ensure that disarticulation of this joint does not take place postoperatively.

Experience has shown that the placement of the prosthesis can be most readily effected in this manner: Once the vein graft is seated over the fenestra ovalis, a probe can be used to tuck the center of the vein section into the fossa ovalis. When this is done, a pocket or invagination results into which the plastic tube can be placed and held upright. A "G" Shea pick is used to press the columella even farther into the fossa ovalis and under the lenticular process. Then the pick is quickly removed and the tube bounces up and fits onto the lenticular projection.

Next, it is necessary to pull the vein graft straight so that it lies flat like a carpet. This action serves to force the prosthesis even farther out of the fossa ovalis, thus fitting it tighter on the lenticular process and producing the proper amount of tension on the vein graft and now reconstituted ossicular chain. If necessary, an angled or oblique Shea pick can be used to fit into the beveled edge of the plastic tube in order to fix it more securely on the lenticular process of the incus.

In order to obtain a properly "tuned" vein graft and prosthesis relationship, the tension and articulation of the system can be tested by applying pressure on the incus to determine if this motion is relayed to the plastic prosthesis and thence to the artificial footplate. If a sufficiently wide excursion of the columella has been obtained, and if this movement is not dissipated because of excessive looseness in the incudo-prosthetic articulation or because the tube section is too short, fluid movements can be seen occurring in opposite phase within the round window niche. In addition, the malleus should be palpated to see if the incus and plastic columella move also.

In addition to testing the efficiency of the columella and substitue footplate, observations regarding changes in auditory acuity can also be made during and after the fenestra ovalis operation. Generally, the patient's hearing is improved when the footplate is removed from the fossa ovalis. When the vein graft is seated, there is a further increase in sensitivity which reaches its maximum as the columella is placed and properly adjusted. When the tympanic membrane is replaced and the canal repacked, the Rinne Test using a 256 cps tuning fork should be performed. A positive Rinne will usually result, in distinction to the negative test result obtained pre-operatively. This finding is a very good indication that a normal or near normal sound energy transmitting mechanism has been achieved as a result of the removal of the otosclerotic lesion and the placement of the prosthesis and vein graft.

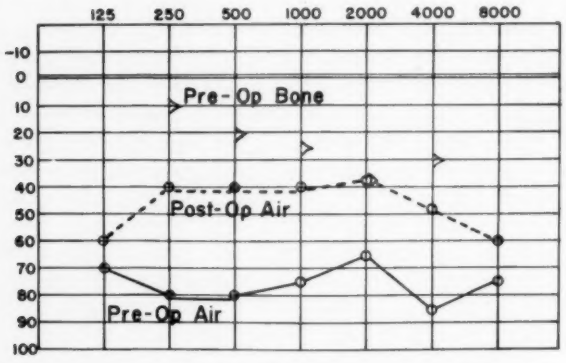


Fig. 4.—Audiometric results following a fenestra ovalis operation in a class C candidate.

Because the fenestra ovalis technique does not involve a mastoidectomy that leaves a large cavity which must heal with a long period of morbidity and draining, the postoperative care of the patient generally is not extensive. If a minimal amount of manipulation has been done in the fossa ovalis, or if suctioning has not removed any perilymph, the patient will experience relatively little surgical reaction. On the other hand, when labyrinthine reactions do appear, they are usually mild in nature and can be controlled by the intravenous administration of Benadryl® and fluids. Patients generally report a feeling of unsteadiness or weaving, rather than a rotatory types of vertigo. Maintaining the patient on Dramamine® 50 mgm three times a day will usually control these sensations.

During this series only four patients experienced a more serious degree of nausea, vomiting and vertigo. The reasons for these increased reactions are obscure, for none of these subjects received surgery more extensive than is usual. They all had periods of unsteadiness and vertigo accompanied by nausea and vomiting with weight loss for about ten days. At no time was there a rise in temperature or nystagmus. It would appear that fenestrating the vestibule produces different reactions than those seen when the lateral semicircular canal is opened.

During convalescence the patient should lie on the unoperated ear for a period of 48 hours until the vein graft has been securely fixed. This precaution also lessens the possibility of labyrinthine

reactions. The patient is also instructed to use only slow, guarded movements for a period of two to three weeks, as this avoids the feeling of giddiness. Most patients are able to eat supper the first postoperative evening and are out of bed for bathroom privileges at the end of 48 hours. The usual hospitalization period is four to five days, and upon returning home there should be no undue activity for seven to ten days. Once the surgical incision has healed, the patient can bathe and swim as before without concern over getting water in the middle ear as is the case with the lateral canal fenestration technique.

## CLINICAL FINDINGS

The audiologic reaction to the fenestra ovalis operation usually follows this pattern: The patient hears very well for a period of 36 hours, at which time the acuity begins to diminish. Hearing tests done on the third or fourth day reveal a great depression of hearing especially in the higher frequencies. This loss in the higher tones appears to be related to the extent of labyrinthine irritation which has resulted from bleeding and aspiration of perilymph. After three weeks have elapsed, the hearing usually begins to show a significant rise in sensitivity. This improvement can be expected to continue until maximal results obtain, and is probably related to the fact that the vein graft becomes firmly seated and more taut. There are exceptions to the above reactions, for some patients show an immediate improvement in aural sensitivity with no sign of auditory regression. It is thought that in these patients trauma to the inner ear is minimal and that the tension upon the vein graft and ossicular chain is optimal from the beginning.

In order to illustrate more fully the scope and effectiveness of this surgical procedure a number of representative cases are presented, along with a description of the pertinent surgical and audiometric findings.

PATIENT NO. 1. This patient, aged 34, presented a history of deafness of many years' duration. Audiometric studies revealed a bilateral conductive hearing loss with a very wide air-bone gap. Because of the nearly normal bone conduction thresholds, this patient falls into the class A category of surgical candidacy. Two previous stapes mobilization operations had been performed with little success, as none of these procedures produced a useful increase in auditory acuity. A Shea fenestra ovalis operation was performed with salutary results (Fig. 2). At the time of operation it was seen that there was a fracture of both crura. Post-surgical audiometry revealed a complete closure of the air-bone gap with an improvement in auditory acuity across the speech range of 43 db. Subsequent hearing tests have shown that this gain in function has been maintained.

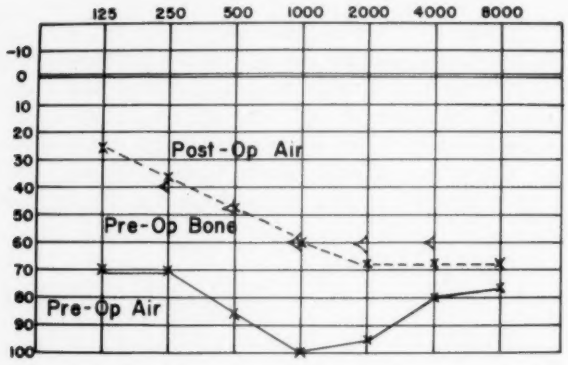


Fig. 5.—Audiometric results following a fenestra ovalis operation in a class D candidate.

PATIENT NO. 2. This patient, aged 50, presented a class B type of surgical problem with a hearing loss of long standing and evidence of some inner ear pathology in addition to a conductive element of 42 db across the speech range (Fig. 3). A successful lateral canal fenestration operation in the right ear had been performed in 1954, while three unsuccessful attempts at stapes mobilization had been made in the left ear. Consequently, a fenestra ovalis operation was done in this ear with excellent results. At the time of surgery a grade IV otosclerosis was found with a diffuse involvement of the entire stapes footplate. Postoperative audiometric study showed that the air-bone gap was almost closed, with the patient showing an average gain in auditory acuity across the speech range of 46 db. Subsequent hearing tests reveal that this improvement has been maintained.

PATIENT NO. 3. This patient, aged 62, had been aware of a hearing loss for 40 years, and presented a severe bilateral mixed conductive deafness, with an average loss by bone conduction of 27 db, which placed her in the class C surgical category (Fig. 4). Previous stapes mobilization surgery had not been successful because of extensive involvement of the stapes footplate. A Shea fenestra ovalis operation was performed with a marked reduction in the air-bone gap. Post-surgical audiometry revealed that the conductive element had been reduced by 27 db across the speech range. While this patient could not get along without a hearing aid before the operation, she was able to engage in most activities after the operation without it.

PATIENT NO. 4. This patient possessed a very severe mixed hearing loss of many years' standing (Fig. 5). Because of the severity of the hearing loss, she was unable to use a hearing aid. The loss by bone conduction in the operated ear was 55 db across the speech range, placing this patient in the class D surgical category. A Shea fenestra ovalis operation was done, and the air-bone gap was almost completely closed. As a result, this patient is now able to use a hearing aid successfully with none of the problems of acoustic feedback and distortion which she faced pre-surgically.

PATIENT NO. 5. This patient is included in this discussion in order to present the type of case in which little or no change in hearing acuity results from the

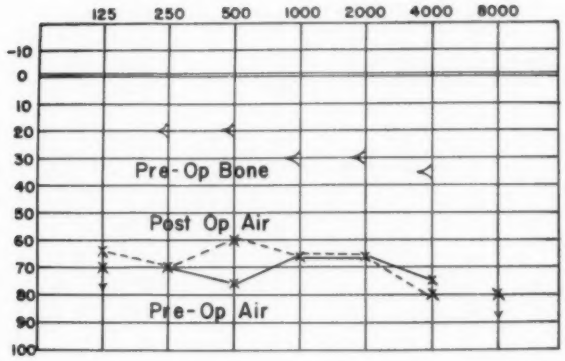


Fig. 6.—Audiometric results of an unsuccessful fenestra ovalis operation in a class C candidate.

fenestra ovalis technique. In this case there had been several previous stapes mobilization attempts. At no time was there a significant increase in auditory acuity. Because of this, it was decided to fenestrate the footplate. At the time of surgery it was found that the patient had a grade IV otosclerosis and the stapes footplate was covered with adhesions. Reference to Figure 6 will reveal that after the operation the threshold values remained at the pre-operative levels, except at 500 cps. Later audiometric testing has shown that the hearing loss is now stabilized. This type of case of which there were eight, gives rise to speculation regarding the factor or factors which prevent the gain in auditory acuity in the presence of a technically correct surgical restoration of middle ear function. It is possible that the following conditions may bring about this lack of improvement in hearing:

The fenestra is too small because it does not conform to the outlines of the oval window. Before it was seen that burring could be employed to produce a patent window, picks were used to attack the otosclerotic bone. In some instances it was not possible to remove all the foci in this fashion. Now, however, the drill is used in every case and all the lesions are removed very effectively, for the otosclerotic bone appears to be superimposed upon the denser bone of the otic capsule, and is usually soft and friable. Furthermore, there is now no hesitation to use the drill within the borders of the vestibule so that it is possible to enlarge the oval window to its maximum.

A second factor leading to failure may be that the vein graft becomes thick and excessively fibrotic, thus producing a barrier to energy transmission into the vestibule. In order to prelude this possibility, the bone surrounding the oval window is now saucerized on the promontory and facial nerve sides so that a shallow depression rather than a deep cup is produced. It is felt that this, together with thinner vein graft section, will eliminate one possible source of failure by preventing vein edge coaptation.

Finally, it is possible that minute errors in columella placement may result in a cancellation of phase difference between oval and round window membranes. This would again lead to a considerable amount of dissipation of the force at the incudo-

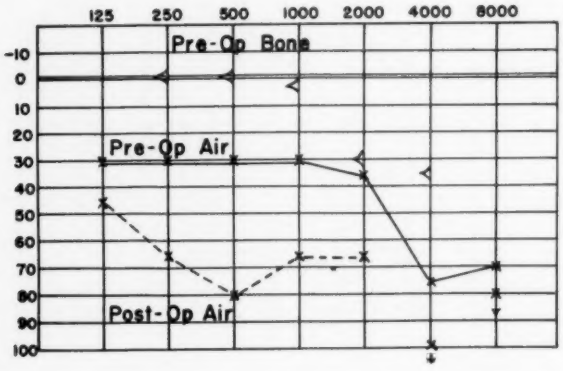


Fig. 7.—Audiometric results of labyrinthitis related to loss of stapes footplate into the vestibule.

prosthetic joint produced by the action of the still intact malleus and incus, with a resultant attenuation of the sound energy reaching the inner ear.

PATIENT NO. 6. As was noted earlier, two stapes footplates have been lost in the vestibule with subsequent loss of cochlear function. Patient No. 6 is an example of this. A right stapes mobilization procedure had been performed with closure of the air-bone gap and normal hearing acuity resulting. Previously, an unsuccessful stapes operation had been done in the left ear. Because we thought that the patient would be benefited by having binaurally symmetrical hearing, it was decided to do a fenestra ovalis operation in the poorer ear. At the time of surgery, a grade IV otosclerosis with crural damage was found. During the operation, as it was attempted to remove the stapes footplate in toto by chiseling it free around the periphery of the fossa ovalis, it fell into the vestibule and could not be recovered. A vein graft with plastic prosthesis was put into place and the drum closed and canal packed in the usual manner. The first postoperative audiogram taken three weeks after surgery revealed a loss in auditory acuity which has increased since then (Fig. 7).

At present, over 100 patients have undergone the fenestra ovalis operation in this department. Of this group, 95 patients have received routine postoperative hearing tests. The results of this audiometric study are most encouraging for they reveal that 69 or 73% of the total group reached or exceeded the 30 db level, while 83 or 87% obtained an improvement of 11 db or more in auditory acuity in the operated ear (Table I). In addition, further analysis shows that 37 or 39% of the patients tested showed a complete or almost complete closure of the air-bone gap, and that 33 or 35% fell within normal limits of hearing (± 15 db) postoperatively.

TABLE I

AUDIOMETRIC RESULTS OF FENESTRA OVALIS OPERATION

| | Early - 3 to 6 weeks | | | | | | | | | Late - 6 to 12 months | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Class | N | 30 db Level or Better | | Gain of 11 db or More | | No gain − 5 to + 10 db | | Loss − 6 db or More | | N | 30 db Level or Better | | Gain of 11 db or More | | No gain − 5 to + 10 db | | Loss − 6 db or More | |
| | | N | % | N | % | N | % | N | % | | N | % | N | % | N | % | N | % |
| A | 60 | 52 | 87 | 55 | 92 | 2 | 3 | 3 | 5 | 15 | 12 | 80 | 14 | 93 | 0 | 0 | 1 | 7 |
| B | 21 | 13 | 62 | 17 | 81 | 3 | 15 | 1 | 4 | 2 | 1 | 50 | 1 | 50 | 0 | 0 | 1 | 50 |
| C | 8 | 4 | 50 | 7 | 88 | 1 | 12 | 0 | 0 | 4 | 1 | 25 | 2 | 50 | 2 | 50 | 0 | 0 |
| D | 6 | 0 | 0 | 4 | 67 | 2 | 33 | 0 | 0 | 3 | 0 | 0 | 3 | 100 | 0 | 0 | 0 | 0 |
| Totals | 95 | 69 | 73 | 83 | 87 | 8 | 9 | 4 | 4 | 24 | 14 | 58 | 20 | 84 | 2 | 8 | 2 | 8 |

In view of the fact that all of the cases reported in this series presented diffuse otosclerosis, the above results compare quite favorably to those achieved by the same surgeons using stapes mobilization techniques. Experience has shown that patients of this type are questionable candidates for the conservative measures provided by stapes mobilization surgery.

Notwithstanding the promising initial results which have been obtained using the fenestra ovalis operation, there are several important questions relating to the fate of the vein graft, the reaction of the middle ear to the plastic tube and the influence upon the cochlea of this technique which must be answered. Therefore, a program of experimental research relating to the fenestra ovalis technique has been started in our otologic research laboratory. The following experimental findings have resulted from this work.

## EXPERIMENTAL FINDINGS

Experiments have been carried out on thirteen cats. The purpose of these experiments was a) to determine by histological methods the fate of the vein graft implanted in the fossa ovalis, and b) to determine how effective the vein graft and columella were in transmitting acoustic energy from the middle ear into the cochlea. This was done by measuring the electrical responses of the cochlea before and after surgical interference.

In each cat, the middle ear was exposed from the ventral side by removing the bone of the tympanic bulla. The stapedius tendon was cut and the stapes removed. A small flap of vein taken from the submaxillary vein was then placed over the oval window after the surrounding area had been previously denuded of mucosa. The vein graft was placed with the adventitious surface lying on the bony wall. Finally, polyethylene tubing (size PE 10), cut to approximately 1.5 mm, was articulated onto the incudostapedial joint at one end, with the other end of the tubing abutting the vein graft at the oval window.

Cochlear microphonics were recorded by means of silver wire electrodes placed on the walls of the round window. The potentials were amplified by a Grass P-4A pre-amplifier, and thence led to a Tektronix 502 oscilloscope. In two cats, silver wire electrodes were implanted in the middle turn of the cochlea and led from there to the skin. These animals were allowed to survive several days postoperatively and recordings were taken during this period from the unanesthetized animals.

In five cats which survived for periods from eight to 106 days, the vein grafts were removed after death, and serially sectioned at 7 μ thickness. The specimens were stained with Hematoxylin and Eosin.

*Fate of the Vein Graft.* On gross post-mortem examination, the adventitial surface of the vein graft had adhered to the bone on which it lay. Blood vessels could be seen entering the tissue, which was viable, and which now appeared to be continuous with the mucosal lining of the middle ear cavity on either side of it, and also with the mucosa overlying the tensor tympani muscle. The columella itself was usually completely covered with membranous-like tissue.

Microscopically, the sections revealed that the vein graft wall had become very much thicker than it had been when implanted. This was mainly due to the invasion of the muscular wall by collagenous tissue. There were many new capillaries present, and also there was considerable revascularization. The most striking finding was that the intimal (free) surface had become covered with a layer of cuboidal cells (Fig. 8a,b) which may represent an attempt at re-endothelialization or re-epithelization. On the other hand, results from the vein grafts of cats which survived only eight days suggest that these cuboidal cells may be basal in nature, and may differentiate much later. Such cuboidal cells and re-vascularization have also been observed in the vein grafts removed from two patients (Fig. 9a,b).

*Electrophysiological Results.* These results are at present preliminary in nature, but they do show certain interesting features. If, after the removal of the stapes, the vein graft is placed over the oval window without the columella, the amplitudes of the cochlear microphonics are considerably reduced when compared to those recorded from the intact animal. The extent of the reduction in amplitude varies with the tonal frequency, being attenuated approximately 60% from normal values for pure tones up to 1000 cps, and then progressively declining to about 85% from the normal at 10,000 cps. In response to a click stimulus, the mean latency of the neural ($N_1$) response is approximately doubled in value.

When the columella is positioned the amplitudes of the cochlear microphonics for tones up to 1000 cps are immediately restored almost to normal levels. For tones above 1000 cps there is again a progressive decline, but this time to about 45% below normal values at 10,000 cps. This reduction, however, can no longer be noticed about 12 hours postoperatively, at which time the amplitudes of the cochlear micro-

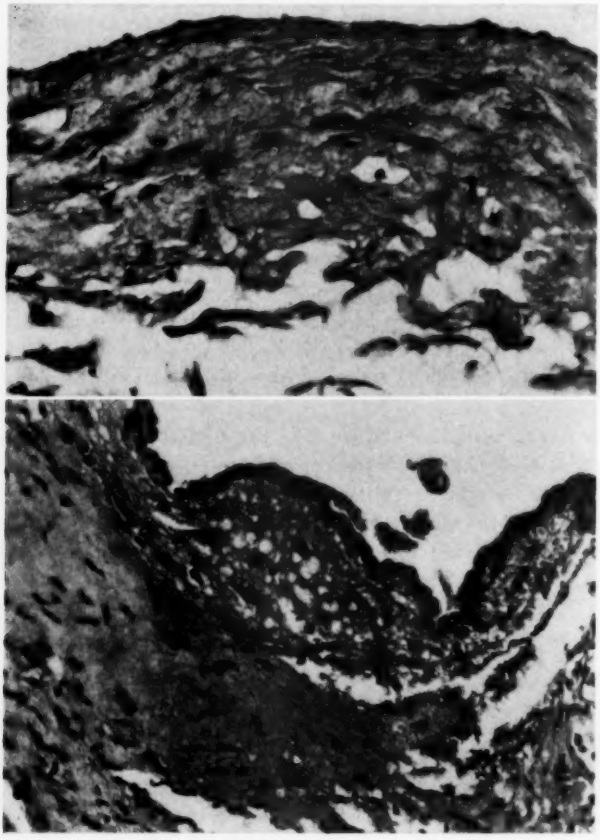

Fig. 8a.—Photomicrograph showing a section of normal submaxillary vein from a cat, 7 μ X 450.

Fig. 8b.—Photomicrograph showing a section of vein graft from middle ear of a cat. Survival period was 48 days, 7 μ X 450.

phonics for all frequencies to 10,000 cps are approximately the same as those recorded from the normal animal. The mean latency of the $N_1$ response to a click stimulus is not significantly different from that of the normal response. If, however, the columella is improperly articulated at the incudostapedial joint, not only are the amplitudes of the cochlear microphonics depressed for all frequencies by about 75% but the mean latency period of the $N_1$ response is increased 2.5

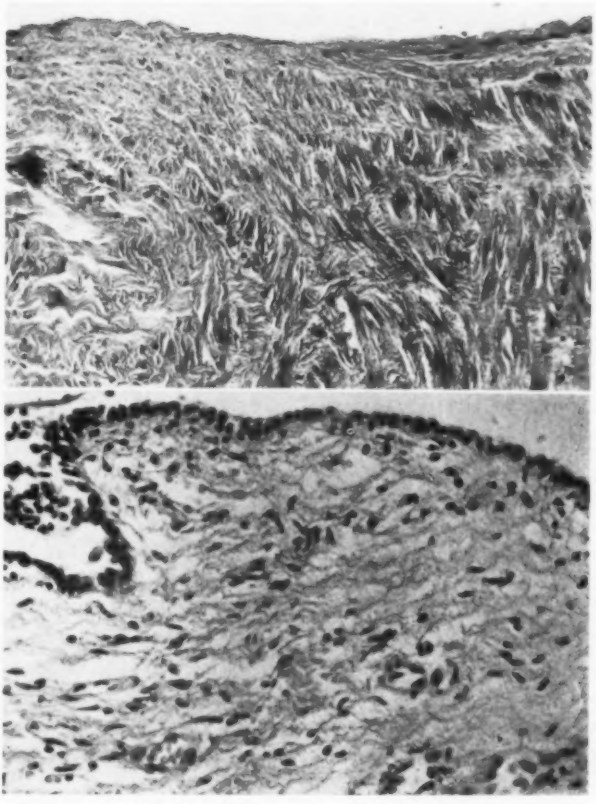

Fig. 9a.—Photomicrograph showing a section from a natural human vein, 7 μ X 225.

Fig. 9b.—Photomicrograph showing a section from a vein graft taken from a patient's middle ear. Survival period was 97 days, 7 μ X 410.

times. This suggests that the improperly placed columella acts as an obstruction to the passage of sound from the middle ear.

It must be emphasized that the results reported above are preliminary, and that further work is now in progress. They do, however, tentatively suggest that under the conditions of these experiments, the vein graft does become viable and that it acts, with the columella, to allow the relatively normal passage of sound from the middle ear to the cochlea.

DISCUSSION

Experience gained in this department during the past year with the fenestra ovalis operation, together with a statistical analysis of our postsurgical hearing tests provides the basis for this discussion of the effectiveness and potential of this important advance in otologic surgery. Although it is still too early to report with finality the value of the fenestration of the footplate in providing long term improvement in auditory acuity for the otosclerotic patient, certain conclusions regarding initially obtained increases in hearing sensitivity as well as an appraisal of patient reaction to this surgery, and postoperative care can be made.

It should be stated that this procedure should not be attempted by the tyro, for it is a difficult technical procedure to master. Only those persons who are well trained in temporal bone surgery and who have had extensive experience with fenestration and stapes mobilization surgery should essay it. Under no circumstances should the fenestra ovalis procedure be attempted without the use of the operating microscope at all operative stages, and without the extensive dissection which is required to produce a clearly visible and easily accessible operating field.

The most recent developments in this technique involving the use of the drill in and around the border of the oval window have been shown to produce a more effective extirpation of the otosclerotic formation than does the use of chisels alone, while at the same time calling for the highest degree of technical skill in creating a patent window into the vestibule. Some otologists have stated that they fear this exposure of the labyrinth. One must hasten to add, however, that the fenestration of the oval window does not appear to be a more radical step than the fenestration of the lateral semi-circular canal. On the contrary, our experience has shown that postsurgical reactions to the former are far milder and of shorter duration than those observed after the latter procedure. It does not seem any more hazardous to place a sterile vein graft over the oval window than setting a skin graft taken from the external auditory canal over the lateral canal fenestration. In addition, postsurgical care following the fenestra ovalis operation has been found to be far less extensive and prolonged than that following the lateral canal fenestration.

The question of auditory results is central to any discussion of the value of otosclerosis surgery, for improvement in hearing is the

ultimate criterion of its success or failure. In this regard, our experience with fenestrating the oval window has led us to conclude that, for the subject with diffuse otosclerosis at least, it has certain advantages over the lateral canal fenestration. In a significant percentage of cases, we were able to produce a complete or almost complete closure of the air-bone gap or hearing acuity which falls within normal limits, results which can rarely, if ever, be attained with the lateral canal fenestration, owing to the fact that the middle ear conductive mechanism is of necessity interrupted, causing an impedance barrier of at least 25 db. It is important at this point to reiterate that the fenestra ovalis operation seeks not only to remove the otosclerotic block to sound energy transmission, but also to re-establish the functional continuity of the sound conducting system.

Although the ideal surgical goal of complete closure of the air-bone gap is initially obtained in approximately one-third of our cases, it is suggested that further refinements in technique and patient selection should increase this proportion. We plan, therefore, to continue our program of clinical and laboratory research in order to bring about a greater understanding of still unsolved surgical problems and the means of dealing with them.

Another area which still requires further study concerns the fate of the vein graft in the middle ear, as well as any possible tissue reaction to the plastic prosthesis. Although at present our data in this regard can be considered preliminary in nature, it appears that the vein graft does take in the majority of patients. Furthermore, because it is autochthonous tissue, it does not seem to set up undue vestibular reactions and does provide a firm, secure and flexible covering for the oval window. In addition, the presence of a layer of cuboidal cells on the free surface of the vein graft suggests that there is an attempt at re-endothelialization or re-epithelialization.

The question of possible foreign body reactions to the plastic prosthesis must be considered. Again, our findings on this score are not conclusive, but they do indicate that there is little, if any, undesirable reaction to the plastic. In the case of the experimental animals, and in the patients who were revised it was found that both the vein graft and the plastic columella were covered with mucous membrane which was continuous with the mucosa lining the tympanic cavity. In addition, it was seen that this membrane acts to tie the prosthesis to the incus, thus increasing the stability of the new incudoprosthetic joint. These findings suggest that there is little or no tendency for the body to reject the plastic prosthesis.

SUMMARY

A preliminary clinical and experimental report of experiences with the fenestra ovalis operation is presented. It is concluded that, for the patient with diffuse otosclerosis at least, this operation provides the otologic surgeon with a more effective means of improving auditory acuity than other stapes mobilization procedures. In addition, it was shown that postsurgical reactions and care are milder and less extensive with the fenestra ovalis operation than with the lateral canal fenestration procedure. Continued research is being carried out in an effort to increase the present proportion of otosclerotic patients given normal hearing or closure of the air-bone gap.

3701 N. Broad St.

We wish to express our appreciation to Drs. L. B. Walker and A. Peale for their courtesy in providing the histological material.

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LXXI

# MIDDLE EAR EFFUSIONS PRODUCED EXPERIMENTALLY IN DOGS:

## I.
## MICROSCOPIC AND BACTERIOLOGIC FINDINGS

JACOB SADE, M.D.
CHARLES D. CARR, M.T.
BEN H. SENTURIA, M.D.
ST. LOUIS, MO.

Middle ear effusions occur spontaneously in humans, often without explanation. In an effort to understand the pathogenesis of these effusions, bacteriologic, cytologic and biochemical examinations were made of such fluids obtained from humans with tubo-tympanitis.[1] As a further means of investigating the mechanism by which effusions occur, experiments were undertaken to produce effusions in animals. Specimens of these fluids were collected for studies and analyses, so that comparison might be made between the characteristics of the human and animal effusions.

This first report of the animal investigations deals with the design of the experiment, the method of producing middle ear effusions in dogs, and cytologic and bacteriologic findings. Biochemical data and histopathologic observations of the involved tissues of these dogs will be reported separately.[2,3]

Beck[4] described his attempts to produce complete atresia of the eustachian tube in dogs. Initially, wooden pegs were inserted into the tube openings, but as these were expelled in a short time, he subsequently applied trichloroacetic acid and galvano-cautery. Many of

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From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri.

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the bullae were found to be filled with purulent secretion which was bacteriologically sterile. These experiments were not considered by the author to be successful, since pathologic examination revealed that total atresia had not occurred in any of the eustachian tubes.

Claus[5] tried to obtain tubal closure by interfering in various ways with the function of the tensor tympani, i.e., by removing the muscle from its attachment to the hamulus, severing its tendon, and injecting paraffin around the tube. None of these procedures was effective. He also split the palate of the animals, exposed the tubal opening and raised various forms of mucosal flaps from the neighboring pharyngeal wall to seal the nasopharyngeal orifice of the tube. In several dogs this latter procedure resulted in an accumulation of fluid in the middle ear which was sterile, but rich in cellular elements. The author concluded that *hydrops ex vacuo* could not be produced experimentally and he considered the effusions which he obtained to be of an inflammatory nature.

G. Holmgren also utilized a mucosal-pouch technique in dogs.[6] A circular incision was made several millimeters away from the margin of the tubal opening. The mucosa between the incision and the tubal orifice was elevated and a ligature was placed around the mucosal neck. Although the number of animals used was not reported, it was stated that fluid was consequently produced in the ear. No information was given about the cytologic components of this fluid. The author also introduced a needle, connected to a manometer, into the bullae and claimed that negative pressures were recorded. It was concluded that this evidence supported the theory of *hydrops ex vacuo.*

L. Holmgren[7] repeated G. Holmgren's experiments and reported his findings in 22 dogs. Four ears contained pus and 12 revealed "no contents" in the bullae. Six ears developed a negative pressure and a small quantity of transudative fluid which was described as "yellowish and a little thick." Microscopic examination revealed "disconnected cell elements and an abundance of mucine fibres."

There followed an active controversy between L. Holmgren[8] and Blegvad[9] concerning the results of these experiments and the conclusions derived from them. The latter apparently not only disagreed with the theory of *hydrops ex vacuo* postulated by the Holmgrens, but questioned the significance of the negative pressure which was measured. Blegvad insisted that the experimentally produced effusions were of an inflammatory origin because of the characteristics of the fluid which was recovered and his belief that any operation on the

eustachian tube would give rise to an inflammatory reaction. He also felt that the relatively long postcautery periods before the effusions appeared made it unlikely that the negative pressure was the cause of the effusions.

The controversy over the animal experiments has its counterpart in the reports dealing with human middle ear effusions; on the one hand are found the cases of classical suppurative otitis media which are recognized as inflammatory in origin and, on the other hand, there is the condition most frequently referred to as serous otitis media which is considered non-inflammatory.

Löwy[10] reported his findings in the fluids obtained from 60 patients with so-called serous otitis media. Forty-two specimens were cultured, of which 38 were sterile and four were considered to be contaminated. All of the examined middle ear specimens contained cells, most of them up to 1000/mm$^3$ with the polymorphonuclears predominating.

Forschner[11] did bacteriological examinations of 40 specimens of serous otitis and found most of them to be sterile. The absence of positive bacterial cultures was attributed to the hypovirulence of the bacteria. He also reported a predominance of polymorphonuclear cells. Forschner observed that most of the patients developed the effusion after an inflammatory process in the nasopharynx and suggested that the fluid in the middle ear was of an inflammatory nature.

Recently, Senturia et al.[1] classified 150 specimens of human middle ear effusions on the basis of the microscopic findings as follows: serous, containing few if any cells; purulent, rich in cells, particularly polymorphonuclears; mucopurulent, containing cells, cellular remnants and mucus; mucoid, containing no cells but myriads of mucous strands. One hundred and thirty bacteriological cultures and 143 smears were examined. Positive cultures were obtained in a large percentage of these specimens and micro-organisms were seen on direct smear.

## METHODS AND TECHNIQUES

The dog was chosen as the most suitable and available experimental animal. Despite a long, tortuous external ear canal, the ear drum can be examined without too much difficulty. It is also possible to visualize the eustachian tube region without splitting the palate.

Six mongrel dogs were used, weighing approximately 25 to 30 pounds. They were vaccinated for rabies and distemper and then

quarantined for at least two weeks prior to the experiment. The animals were anesthetized with intravenous pentobarbital sodium (60 mgm/kg), and 10% cocaine hydrochloride was sprayed into the nasopharynx. The usual time for the animals to awaken from the anesthesia was two to four hours. No untoward effects from the anesthesia were noted.

On the day of cautery, which will be referred to as "zero-day," cultures were obtained from the nasopharynx and the external auditory canals of each dog prior to cauterization of the eustachian tube. Accumulated wax was then removed from the canals with a dull curette and the meatal skin was cleaned with ether-saturated swabs. Subsequently, the external ear canals and nasopharynx were cultured prior to each aspiration of the middle ear cavities. The cotton swabs were inoculated directly onto the surface of blood agar plates for primary identification of bacteria. Supplementary differential studies were performed when indicated.

The cauterization technique employed was demonstrated to us by Dr. G. O. Proud.[12] An 8 mm cautery tip was introduced into the lumen of one eustachian tube for 40 seconds. Then the torus tubaris and 1 to 2 mm of the peritubal area was cauterized. The contralateral tube served as a non-traumatized control.

All middle ears were aspirated on the 3rd, 6th, 9th and 14th days after zero-day. Subsequently, the six dogs were divided into three pairs and sacrificed at planned intervals. The middle ears of the first pair were aspirated on the 18th day and the dogs were then sacrificed. The second and third pairs were aspirated and sacrificed on the 22nd and 26th days (Table I).

Because of the anatomy of the dog middle ear bulla, it was difficult to aspirate all of the fluid from this cavity without traumatizing the middle ear structures. A special aspirator previously described[1] was used for puncturing the tympanic membrane and removing the fluid from the middle ear. This instrument has a double tube arrangement that permits the fluid to be collected into the inner test tube which can be conveniently and aseptically stored without transferring the fluid to another tube.

The aspirated fluid was cultured immediately. Smears for cytologic study were made in duplicate; one was stained with Wright's blood stain for cell differentiation and the other with Gram's stain for identification of bacteria.

TABLE I

TIME OF ASPIRATION OF MIDDLE EARS
AND TYPES OF EFFUSIONS OBTAINED

| Dog Number | | 8 | | 9 | | 10 | | 11 | | 12 | | 13 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ear | | L | R | L | R | L | R | L | R | L | R | L | R |
| Eustachian Tube Cauterized | | + | | + | | + | | + | | | + | | + |
| Interval | 3 | O | O | O | O | O | O | O | O | O | P | O | P |
| (in days) | 6 | P | O | P | O | P | O | O | O | O | P | O | P |
| between | 9 | P | P | P | O | P | O | P | O | O | P | O | O |
| cauterization | 14 | O | P | P | O | O | S | P | O | O | P | O | MP |
| and | 18 | | | | | *O* | *S* | | | | | *O* | *P* |
| aspiration | 22 | O | S | O | O | | | | | | | | |
| | 26 | | | | | | | *P* | O | O | *P* | | |

O = Aspiration performed, but no effusion found.

S = Serous effusion.

P = Purulent effusion.

MP = Mucopurulent effusion.

*Italic* letters indicate time that dog was sacrificed.
For the cytologic characteristics on which classifications were based, see Reference 1.

## RESULTS

*Production of the Effusions* (Table I). All of the middle ears which had their eustachian tube openings cauterized, developed fluid ranging in amounts from one drop to a milliliter. Effusions were found in two ears (12-R and 13-R) on the first aspiration (third day after cauterization). Three ears (8-L, 9-L and 10-L) showed their first effusions on the second aspiration (six days after cauterization). One ear (11-L) contained fluid for the first time on the third aspiration (9th day). One dog showed fluid in the middle ear (12-R) on each of the five aspirations while another (13-R) did so on four occasions. Two middle ears (9-L and 11-L) contained fluid on 3 successive occasions. Fluid was found in the middle ears of two dogs (8-L and 10-L) on two successive aspirations (6th and 9th days) but not thereafter.

The control ear of each dog was aspirated routinely at the same time as the cauterized ear. Four of these control ears remained nega-

tive throughout the experiment while the other two developed fluid at irregular intervals. An effusion was present in one of the controls (8-R) on the third aspiration (9th day) and appeared in this ear twice more. In one other control (10-R) an effusion was present on the fourth aspiration (14th day) and occurred in this ear once more.

*Macroscopic Appearance of the Effusions.* The effusions differed in color and consistency, varying from thin, watery and translucent to viscous, opaque, yellow or gray. There was no clear relationship between the gross appearance of the effusion and the interval between cautery and aspiration. Some fluids appeared serous while others wre purulent on the same postcautery date. In five specimens a small amount of fresh blood appeared in the fluid, usually during the latter part of the aspiration.

*Microscopic Findings in 24 Effusion Specimens*

White Blood Cells:

Polymorphonuclear Neutrophiles:

All the effusions contained polymorphonuclear neutrophiles as the predominant cells, and there was no decrease in their number during the experimental period. The serous specimens contained only a small number of cells and these were mainly neutrophiles. In the first aspirations, the neutrophiles were intact, while in the last specimens aspirated from three dogs, there was suggestive evidence of degeneration, including loss of cell membrane and dissolution of the cytoplasm.

Lymphocytes:

Lymphocytes were present in all of the specimens. They constituted about 5% of the total number of white blood cells and there appeared to be no correlation between the number of lymphocytes and the age of the effusion.

Monocytes:

Monocytes were found in six of the specimens. These appeared in the effusions during the latter part of the experiment and amounted to about 1% of the leucocytes.

Eosinophiles:

No eosinophiles were seen in any of the specimens.

Macrophages:

Macrophages were found in 13 of the specimens, constituting approximately 1% of the cells. Their presence did not seem to be related to the time after cautery that the fluid was aspirated.

Cellular Remnants:

A small number of unidentifiable cell remnants appeared in all of the specimens.* There was no relationship between the number of these remnants and the duration of the effusions.

Mucus:

A slight amount of mucus was observed in 12 of the specimens while numerous mucous strands were noted in one instance.* No correlation could be found between the appearance or quantity of mucus and the time of aspiration.

Red Blood Cells:

All of the specimens contained some red blood cells, but the ratio of red cells to white cells was such as to rule out the presence of any large amount of blood.

*Bacteriologic Findings:*

Bacteria were cultured from 22 of the 24 effusions and included alpha and beta hemolytic and non-hemolytic streptococci, H. influenzae, micrococci and Pseudomonas aeruginosa. In five of the six dogs, swabs of the nasopharynx taken on zero-day revealed six cultures of micro-organisms similar to those which were found in the middle ears of these same dogs. Four of these were micrococci, one a streptococcus and one pseudomonad. In eight instances, exogenous bacteria which first appeared in the nasopharynx after zero-day were found on the same day or later in the middle ears. Three of these were beta-hemolytic Streptococci, one gamma-hemolytic Streptococcus, one a non-hemolytic Streptococcus and three micrococci.

Streptococci were cultured from the external ear at the same time or after their appearance in the middle ear in five instances. Six micrococci and one beta-hemolytic Streptococcus were cultured from the external ear canal on zero-day and were found later in the middle

*One specimen was highly viscid and contained numerous cellular remnants and mucous strands. On the basis of the suggested classification it was designated as mucopurulent.

ear specimens. In one more case, micrococci which appeared in the external ear after zero-day were later found in the middle ear.

Seventeen of the 24 effusions aspirated from the middle ears showed intracellular or extracellular bacteria or both on direct smear. Organisms were present as early as the 3rd day in one specimen and in all except one on the 6th and 9th days. From the 14th day on, approximately one-half of the specimens showed bacteria on smear. In addition, these same ears showed positive cultures on these days.

## COMMENT

This study confirms the fact that it is possible to produce middle ear effusions fairly consistently in the dog by cauterizing the nasopharyngeal orifice of the eustachian tube and the immediate peritubal area.

The evidence presented here corroborates the observations that traumatization of the nasopharyngeal orifice of the eustachian tube produces an inflammatory type of effusion similar to those described by Beck and Claus. Even those fluids which appear macroscopically to be of a "transudative type," and might be interpreted as resulting from a *hydrops ex vacuo*, reveal on microscopic examination the purulent nature of the effusions.

The cytologic pictures of all effusions show typical purulent characteristics in the predominance of neutrophiles and the relatively small number of lymphocytes and monocytes. During the 26 days of the experiment there was no decrease in the number of neutrophiles nor any increase in the percentage of lymphocytes, monocytes or macrophages. The absence of eosinophiles was noteworthy.

Cellular remnants were seen in all the effusions in small numbers, and never occurred to the degree seen in human material. There was some indication in the specimens obtained in the latter part of the experiment that degeneration of the neutrophiles was taking place, but the observations were not extended over a sufficiently long period to be conclusive.

It is assumed that the disease produced by cautery of the nasopharyngeal orifice and peritubal area resulted in a nasopharyngitis, salpingitis and otitis media. The presence of effusions in two of the control ears is attributed to the extensive nasopharyngitis which developed following cautery.

As a consequence of the bacterial contamination of the middle ear, there was a continuing accumulation of fluid in the lumen and a persistent migration of new neutrophiles into the effusion. Just as in the report of findings in the human,[1] a high percentage of the middle ear specimens obtained in this animal experiment showed bacteria on culture and smear. The positive cultures and presence of bacteria on smear confirm the purulent nature of the effusions.

The source of the bacteria in these specimens was sought in the nasopharynx or the external ear. In many instances, a given strain of bacteria was found in the nasopharynx prior to or on the same day that it was recovered in the middle ear effusion, but in only half of the cases were these bacteria recovered from the nasopharynx before cauterization of the eustachian tube. When an exogenous strain of bacteria appeared in the nasopharynx after cautery and was cultured later from the middle ear, it is assumed that infection was superimposed in the nasopharynx and then ascended the eustachian tube into the middle ear.

On six occasions, the appearance of a micrococcus in the middle ear was preceded by its discovery in the corresponding external ear canal. Consideration must be given to the possibility that the organisms, in these instances, were carried into the tympanum from the external ear with the aspirating needle. It is our impression that during aspiration of a healthy middle ear, contamination with endogenous external ear organisms does not readily occur. However, in those instances where disease of the nasopharynx is present and eustachian tube function is interfered with, the middle ear may be more susceptible to contamination from the external ear.

In some instances the positive cultures in the external ear appeared simultaneously with or after the appearance of a similar strain in the middle ear. This suggests that perforation of the tympanic membrane permitted drainage of the purulent middle ear fluid into the ear canal and thereby accounted for the contamination of the meatus.

When the gross characteristics of the fluids obtained from the middle ears were observed, it was striking to note the great variability of appearances and consistency. The specimens ranged from watery transparency to a thick and mucoid opacity. In human beings we also found effusions varying greatly in physical properties. Chemical analyses of the specimens help to partially explain these great differences in viscosity. Some aspects of this problem were previously

explored in the report on human specimens[1] and others regarding these dog effusions are discussed in a concurrent publication.[2]

Popular opinion holds that effusions of recent origin in humans are serous and later become gluey and thick. This viewpoint was not supported by the observation on our limited number of specimens since the characteristics of the fluid were unrelated to the interval between cautery and aspiration.

SUMMARY

Experimental middle ear effusions were produced by cauterizing the nasopharyngeal orifice of the eustachian tube in the dog.

The large majority of experimental effusions had microscopic characteristics similar to those obtained in spontaneous human purulent otitis media. A small number resembled specimens of serous otitis media.

Positive bacterial cultures were obtained in almost all of the middle ear specimens and intracellular or extracellular bacteria or both were seen on direct smear in more than half of the fluids.

The physical and cytologic characteristics of the experimental effusions were not found to correlate with the length of time that the effusions were present in the middle ear.

500 N. Skinker Blvd.

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LXXII

# MIDDLE EAR EFFUSIONS PRODUCED EXPERIMENTALLY IN DOGS:

## II.
## SOME BIOCHEMICAL AND ELECTROPHORETIC PROPERTIES

CARL F. GESSERT, PH.D.

E. S. BAUMANN, B.A.

BEN H. SENTURIA, M.D.

ST. LOUIS, MO.

Middle ear effusions were produced experimentally in dogs, and the microscopic and bacteriologic findings concerning them have been reported.[1] Certain biochemical analyses were made on these effusions, primarily so that their characteristics could be compared with those presented previously for effusions which occur spontaneously in the human middle ear.[2] A secondary objective of the analyses herein reported was to determine whether there was a similarity in composition between the fluids obtained from the ears and the blood sera of the same animals.

### METHODS AND PROCEDURES

The experimental middle ear effusions were produced in six dogs by electrocautery of the nasopharyngeal orifices of the eustachian tubes. At various intervals after cautery, specimens of the effusions were collected for analysis.[1] Near the end of the experimental period, blood samples were taken from these dogs and from eight control dogs, which received no cautery nor other treatment so that similar analyses could be made on the sera.

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From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri.

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The details of the methods of producing and collecting effusions have been presented.[1] In each experimental dog, one eustachian tube orifice was electrically cauterized while that of the contralateral tube was not. Periodically thereafter, the tympanic cavities of both ears were aspirated, regardless of whether or not an effusion was known to be present. When a specimen was obtained, a small portion of it was examined cytologically and then classified on this basis as serous, purulent or mucopurulent, by previously established criteria.[2]

Determinations were made of the percentage of dry matter, total protein, deoxyribonucleic acid (DNA) and protein-bound carbohydrate in the whole effusion specimens and in the sera of both experimental and control dogs. The methods of analyses were the same as those reported previously,[2] except that instead of 6% perchloric acid, ethanol was used to precipitate the proteins (24 parts absolute ethanol to 1 part specimen). The averages for the various categories were then compared with each other and with the values previously obtained for human middle ear effusions.[2]

The protein fractions which were present in the unprecipitated effusion specimens and the sera were examined by means of paper electrophoresis. When an attempt was made to carry out the electrophoresis on whole specimens of the effusions, it was found that there was much insoluble matter which remained at the origin and interfered with the electrophoretic pattern. Consequently, portions of all of the specimens were placed into an ultracentrifuge which was run at 25,000 rpm for one-half hour. The supernatants, without cells and cellular debris, were then subjected to electrophoresis on paper strips, and similarly treated sera of the experimental dogs, as well as untreated sera of control dogs, were run for comparison.

The method used for electrophoresis was one which employed Spinco cells and the Model RA Analytrol.[3] The paper strips were Whatman 3 MM and the buffer was composed of barbituric acid and barbiturate at pH 8.6, ionic strength 0.075. The voltage was regulated so that a constant current of 5 ma. per cell of eight strips was maintained. The electrophoreses were run for 16 hours in a cold room at 4° C., rather than at room temperature, in an effort to increase stability and resolution.[4] The heat-fixed protein fractions were stained with bromphenol blue, then the strips were run through the Analytrol. One group of strips was analyzed also by eluting the bromphenol blue-stained protein fractions separately with sodium carbonate solution and determining concentrations colorimetrically. The net results were not significantly different from those obtained with the Analytrol.

TABLE I

CERTAIN COMPONENTS IN DOG MIDDLE EAR EFFUSIONS AND SERA

| | TYPE OF SPECIMEN | NO.* | PROTEIN | DNA | PROTEIN-BOUND CARBOHYDRATE | DRY MATTER |
|---|---|---|---|---|---|---|
| | | | *As Mean Percentages of Whole Specimens* | | | |
| Effusions: | Serous | 2 | 9.49 | 0.033 | 0.069 | 12.56 |
| | Purulent | 18 | 6.84 | 0.228 | 0.128 | 10.72 |
| | Mucopurulent | 1 | 9.17 | 0.291 | 0.107 | 13.4 |
| Sera: | Experimental | 6 | 6.09 | 0.0047 | 0.0405 | 8.66 |
| | Control | 8 | 5.93 | 0.0050 | 0.0420 | 8.33 |
| | | | *As Mean Percentages of Dry Matter* | | | |
| Effusions: | Serous | 2 | 75.4 | 0.263 | 0.550 | |
| | Purulent | 18 | 65.0 | 2.125 | 1.183 | |
| | Mucopurulent | 1 | 68.5 | 2.17 | 0.800 | |
| Sera: | Experimental | 6 | 70.4 | 0.054 | 0.468 | |
| | Control | 8 | 71.1 | 0.060 | 0.504 | |

*One of the serous and two of the purulent specimens were of insufficient quantity to perform all of the analyses, so they were omitted from the averages.

## RESULTS

The schedule of aspirations, the findings regarding the presence or absence of effusions, and the cytological classification of the effusions are all shown in a concurrent publication.[1] In four dogs the effusions developed only in the ears of cauterized eustachian tube orifices, but in the other two dogs, effusions were found in both ears. Two purulent and three serous effusions were obtained from control, uncauterized ears, while 18 purulent specimens and one mucopurulent were taken from ears whose eustachian tube orifices had been cauterized.

Results of the analyses of whole specimens for dry matter, total protein, DNA and protein-bound carbohydrate are shown in Table I. Averages are presented for the various categories of experimentally produced ear effusions and for the sera of the experimental and control dogs. In Table II there are presented for comparison the averages of similar data obtained previously on human effusions and normal sera.[2]

TABLE II*

CERTAIN COMPONENTS IN HUMAN MIDDLE EAR EFFUSIONS AND SERA

| | TYPE OF SPECIMEN | NO. | PROTEIN | DNA | PROTEIN-BOUND CARBOHYDRATE | DRY MATTER AS MEAN PERCENTAGE OF WHOLE SPECIMEN |
|---|---|---|---|---|---|---|
| | | | *(As mean percentages of Dry Matter)* | | | |
| Effusions: | Serous | 13 | 73.5 | 0.05 | 0.53 | 15.5 |
| | Purulent | 10 | 64.4 | 1.52 | 0.79 | 11.8 |
| | Mucopurulent | 10 | 66.1 | 0.77 | 3.07 | 16.8 |
| Sera: | Control | 4 | 66.5 | 0.01 | 0.35 | 9.4 |

*Data taken from Tables VI and VII of Reference 2.

The data on dog specimens revealed that, on the basis of wet weight, all categories of effusions contained higher percentages of the various components under investigation than the sera. This was also true on the dry weight basis, except for total protein in purulent and mucopurulent effusions which were slightly lower. The purulent effusions, which contained large numbers of cells, were much higher in DNA content than the serous effusions and sera. Even though the serous effusions contained only about an eighth as much DNA as purulent specimens, they were still considerably higher in DNA than sera. The serous effusions contained slightly more protein-bound carbohydrate than the sera, whereas the purulent effusions contained over twice as much.

In general, the percentages of the above components in the dry matter of experimentally produced middle ear effusions of dogs were a little higher than those in the corresponding categories of human specimens (Tables I and II). The one mucopurulent dog specimen was an exception in that it contained a much lower percentage of protein-bound carbohydrate than the average for human specimens.

The averages of these same components in the blood sera of experimental dogs did not differ significantly from those of control dogs.

Paper electrophoreses were run on the control dog sera and on supernatants of the ultracentrifuged portions of all experimental

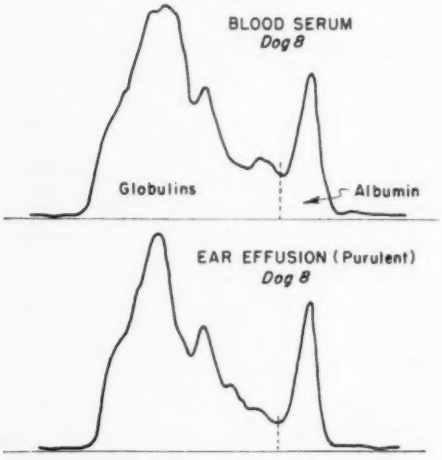


Fig. 1.—Electrophoretic Patterns of Protein Fractions. The electrophoretic patterns were obtained by running the paper strips through the Analytrol after the electrophoreses were carried out on centrifuged specimens. The middle ear effusion was a purulent one obtained from the left ear, nine days after cautery.

dog sera and effusions. In the blood sera of both the experimental and control dogs, the electrophoreses showed a much lower proportion of albumin and higher proportions of globulins than were found in normal human blood serum proteins run under the same electrophoretic conditions. However, when the sera of the experimental dogs were compared with those of the untreated control dogs, there were no obvious differences between their respective protein patterns. The albumin constituted an average of 28.7% in the untreated and 26.0% in the experimental dogs.

Electrophoresis of the effusion specimen supernatants showed that they also contained high ratios of globulin to albumin, similar to those found in the blood sera of these dogs. The purulent effusions were especially high in globulins, yet there seemed to be no distinctive features about the patterns which would differentiate one effusion from another, or even from a sample of serum. A striking similarity is shown in Figure 1 between the electrophoretic patterns obtained from the centrifuged serum of one dog (No. 8) and a centrifuged, purulent effusion specimen aspirated from the left ear of the same dog on the ninth day after cautery of the eustachian tube orifice. The

$\alpha_2$-globulin fraction seemed to be rather high in some of the effusions, and that fraction interpreted to be $\beta$-globulin was excessively high in specimens which contained hemolyzed red cells.

The concentrations of protein were measured in the supernatants of the ultracentrifuged samples of sera and effusions in order to compare them with the concentrations in the whole specimens. In purulent effusions there was an average of 85% as much protein in the supernatants as in the whole specimens, whereas in sera and serous effusions the "supernatants" contained 99% as much protein as the whole specimens.

Analyses were also made of DNA and protein-bound carbohydrate in the supernatants of ultracentrifuged portions of six of the purulent specimens. In these samples, the concentration of DNA in the supernatants averaged only 11% of that in the same six whole specimens, whereas the protein-bound carbohydrate averaged 59%.

## COMMENT

Possible changes were sought in the compositions of the effusions with respect to the interval between cauterization and aspiration, but the observations were not conclusive. There seemed to be some tendency for protein and dry matter to be slightly higher in effusions taken after longer intervals, but this pattern was not evident in all ears. The interpretation of changes with time would be difficult in any case because it is not known how completely the effusion is removed by aspiration at any given time. It is likely that each successive specimen from the same ear is composed partly of newly formed effusion and partly of accumulated effusion which was not completely removed by previous aspiration.

The fact that most (20 of 24) of these experimentally induced effusions were purulent, rather than serous, indicates that the mechanism of their production was one of inflammatory reaction, rather than a *hydrops ex vacuo*. The chemical finding of a high DNA content in the purulent specimens bore out the cellular and inflammatory nature of these effusions. Even the serous specimens contained enough DNA to indicate that they were not purely transudates of blood plasma, such as one might expect in a *hydrops ex vacuo*.

The results of the analyses of the supernatants indicate that about 90% of the DNA and some 40% of the protein-bound carbohydrate in purulent effusions can be removed by ultracentrifugation.

When cells and cellular material were thus removed, the remaining fluid contained concentrations of DNA and protein-bound carbohydrate which were very close to those of serous effusions. It seems, therefore, that the presence of cells and cellular matter is chiefly responsible for these chemical differences between purulent and serous effusions. It is not clear, however, why the serous effusions, both whole specimens and supernatants alike, were higher in total protein content than whole, purulent effusions and their supernatants.

From the paper electrophoreses findings, there were no consistent differences seen in the protein patterns obtained from the supernatants of purulent or serous effusions and sera. It was not easy, nor in many instances even possible, to distinguish between effusion specimens and serum, especially from the same dog, on the basis of the protein patterns found. In view of the similar electrophoresis patterns and the other chemical findings in serous specimens and purulent specimens from which the cells have been removed, it seems likely that the major part of the noncellular portions of these two types of effusions have a common origin, i.e., blood plasma.

It is of interest that only one specimen obtained in this series of experimental effusions was classified as mucopurulent, whereas the mucopurulent category constituted about half of the total of spontaneous human middle ear effusions.[2] It would appear that mucopurulent effusions occur when there is a smoldering inflammation which stimulates a metaplasia of the mucoperiosteum of the middle ear and hyperactivity of the glands in the epithelium lining of the tympanic orifice of the eustachian tube. Mucus which would be produced by these glands could account for the high content of protein-bound carbohydrate in mucopurulent effusions and could be responsible for their high viscosity. Another possible source of the glycoprotein (protein with a high proportion of carbohydrate bound to it) is the ground substance of the mucoperiosteal lining.

Whatever the source may be, it appears that most of these dog middle ears had not reached a stage in which there was sufficient glycoprotein present to give the fluid the physical characteristics of a typical mucopurulent effusion. It was our impression, however, that many of the purulent dog fluids were more viscous than the human purulent specimens. This apparent tendency toward mucopurulent properties is not surprising in view of the fact that the average protein-bound carbohydrate content and also the DNA content of dog purulent specimens were somewhat higher (approximately 40 to 50%) than the corresponding human specimens.

SUMMARY

Effusions were experimentally produced in the middle ears of dogs by cauterizing the nasopharyngeal orifices of the eustachian tubes. These effusions were collected from the tympana by aspiration at various intervals. In a period of about three to four weeks, 24 specimens were collected from six dogs. Twenty of these were classified as purulent, one as mucopurulent and three as serous.

Analyses were performed on specimens of effusions, and on blood sera which were obtained from experimental and control dogs. Determinations of protein, dry matter, deoxyribonucleic acid, and protein-bound carbohydrate showed that the concentrations of these components in serous and purulent specimens were generally a little higher than, but still within the same range of those of corresponding categories of spontaneous human effusions. The concentration of DNA in purulent effusions was about seven times as high as that in serous effusions and that of the protein-bound carbohydrate was about twice as high. Nevertheless, when cells and cellular debris were removed from the purulent effusions by ultracentrifugation, the supernatants had about the same concentrations of these components as serous effusions.

Paper electrophoresis of the supernatants of all effusions and blood sera indicated that there were no consistent differences by which they could be distinguished from one another.

There was no relationship found between changes in the composition of the effusions and the interval between cautery of the tubes and aspiration of the middle ears.

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The authors wish to express their appreciation to Dr. Oliver H. Lowry for his consultation and for making available laboratory facilities in the Department of Pharmacology, and to Mr. Charles D. Carr and Dr. Jacob Sade for their co-operation in conducting the experimental study.

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LXXIII

# THE EFFECTS OF TOPICAL ANESTHETICS UPON THE EAR

W. E. Rahm, Jr. W. F. Strother
W. L. Gulick J. F. Crump
Princeton, N. J.

According to the electrical theory of auditory nerve stimulation the nerve impulses are triggered by the cochlear response, a small potential generated by the hair cells of the cochlea during auditory stimulation. It is apparent, therefore, that any factors which adversely affect the cochlear response also produce deleterious effects upon hearing, since hearing can be no better than the cochlear potential allows.

Topical anesthesia is considered by many surgeons to be the method of choice for operations on the ear. If this type of anesthesia is properly employed there will be adequate relaxation, control of bleeding, a margin of safety for the patient, and the co-operation of a conscious patient.[9] However, evidence suggests that certain currently used topical anesthetics produce irreversible losses in the cochlear response when these solutions are employed to anesthetize middle ear structures during surgery. Certain other anesthetics apparently produce little or no change in the cochlear response.

It is generally known that certain chemical substances depress the actions of living cells. Accordingly, experimental studies of the electrical response of the cochlea have used chemical agents to determine whether or not the cochlear response is a physiological potential or nothing more than an artifact. Chemicals were first applied directly to the cochlea by Adrian, Bronk and Phillips.[1] In their investigation of the source and nature of the electrical response of the cochlea they injected cocaine solutions through the round window membrane. They observed that the cochlear response diminished rapidly. As a control solution they employed physiological saline,

From the Princeton Psychological Laboratory. This work was supported by a contract between the Office of Naval Research and Princeton University Nonr 1858(18), and by Higgins funds allotted to Princeton University. Permission is granted for reproduction and use by the United States Government.

and observed that it had little effect upon the magnitude of the response. Guttman,[7] following the same procedure of application, found that the response was abolished by cocaine, quinine, salicyl, and pilocarpine solutions. The response was not affected by applications of saline, camphor, and strychnine solutions. Davis, Derbyshire, Lurie, and Saul[3] placed novocaine crystals (procaine hydrochloride) upon the surface of the round window membrane and thereby abolished the cochlear response. Their procedure insured that the loss of the cochlear response was not the result of mechanical damage to the cochlea during the injection of solutions. Hallpike and Rawdon-Smith[8] reported a rapid and steady decline in the magnitude of the cochlear response following the application of either cocaine or sodium chloride crystals to the round window membrane.

More direct investigations of the effects of chemical substances upon the cochlear response have been pursued by Fowler and Forbes.[4-6] No losses in the response recorded on dogs and cats were observed following the application of distilled water, glucose, or dextrose to the round window membrane, and histological examinations revealed no cellular damage. However, these investigators reported a progressive diminution in the electrical response of the cochlea following application of glycerin, sodium chloride, calcium chloride, and quinine di-hydrochloride to the round window membrane. Whenever these substances were applied in quantity for an extended period of time the response was abolished. Furthermore, there was little behavioral evidence of recovery of the response for periods up to 25 days. Histological examinations of the area revealed marked destruction of the hair cells and contiguous tissue.

Wever and Bray[11] placed sodium chloride crystals upon the round window membrane of the guinea pig. Their data agree with those obtained by other investigators. The reduction of the response was a function of the quantity of sodium chloride applied. Losses ranged from 10 to 50 db below the output obtained prior to the application of the crystals. Any recovery noted was of a temporary nature and it was followed by further decline.

The present experiment studied the effects of cocaine in isotonic saline and hexylcaine hydrochloride isotonic (Cyclaine) topical solutions on the cochlear response of the cat.

## PROCEDURE

Each animal was anesthetized with a solution of diallyl-barbituric acid and ethyl carbamate injected intraperitoneally in a dosage of 1 cc

per kilogram of body weight. After a surgical level of anesthesia had been reached the tympanic bulla was approached laterally through an oval incision in the skin covering the parotid gland. Removal of this gland and resection of the sternocleidomastoid, digastric, and stylohyoid muscles exposed the bulla.

Two holes were drilled in the lateral surface of the bulla. One hole (diameter 3 mm) was located near the stylomastoid foramen to accommodate the wire from the active electrode which terminated on a piece of platinum foil placed upon the round window membrane. A second hole (diameter 6 mm), located in an anterior position toward the jugular foramen, was used initially for purposes of inspection during electrode placement and thereafter to gain access to the round window membrane for the application and removal of various anesthetic substances.

A tonal stimulus of 1000 cps was generated by a General Radio Type 913-C beat-frequency oscillator whose output was connected to a Daven Type T-693 attenuator. The output from the attenuator fed into a power amplifier which drove a Western Electric 555 receiver. A hard rubber tube which terminated in a cannula tied into the external meatus led aerial sound from the speaker to the tympanic membrane. Cochlear potentials picked up from the round window membrane were amplified and measured with a General Radio Type 736-A wave analyzer operated as a selective voltmeter.

The intensity of the tonal stimulus was adjusted initially so as to give a cochlear response of 50 microvolts. After the stability of the response was assured, one of several anesthetic substances was applied to the round window membrane by means of a small nylon tube affixed to a hypodermic needle on a 1 cc syringe. The blunt end of the tube, placed in the bulla cavity through the larger hole, was located directly over the round window membrane at a distance of about 2 mm. The dosage of anesthetic substances was always 0.25 cc. After fifteen minutes the anesthetic was removed by using the nylon tube as a catheter. In addition, the membrane was dried with a cotton wick. A previous study[10] shows that long term exposure of the middle ear cavity does not significantly change the cochlear potential.

The effects of the anesthetic upon the cochlear response were evaluated in two ways. First, changes in the magnitude of the response produced by a constant stimulus were observed over extended periods of time. Second, intensity functions were plotted immediately before and after application of anesthetics, as well as at regular intervals following withdrawal.

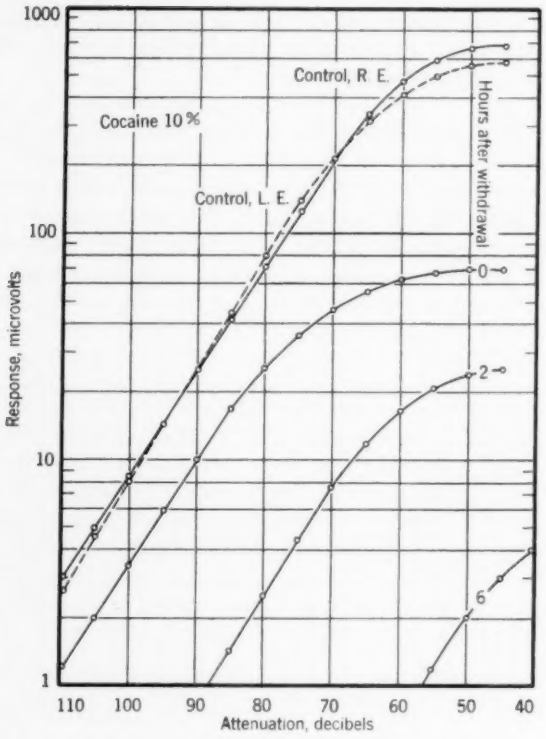


Fig. 1.—Irreversible effects of 10 per cent cocaine upon cochlear potentials.

In this study one control and three experimental solutions were investigated. The control solution consisted of physiological saline, and the experimental solutions consisted of cocaine in saline (10 per cent), and hexylcaine hydrochloride (Cyclaine, Sharp and Dohme) either 5 or 1 per cent solutions. Sixteen animals were employed, four for each of the solutions. The temperature of the solutions upon application equalled body temperature (34° C). All animals were isolated in an electrically shielded soundproof room.

## RESULTS

Application of any of the four solutions to the round window membrane produced an immediate and precipitous drop in the cochlear

response. This drop may be ascribed to the shunting effect of the solutions which acted as electrolytes and effectively short-circuited the response. Further changes in the magnitude of the response following this initial drop depended upon the particular solution applied to the round window.

*Physiological Saline.* When the solution was withdrawn and the round window membrane dried, the response recovered fully without any latency and remained stable as long as the preparation remained in satisfactory condition.

*Cocaine in Saline* (10 per cent). Figure 1 presents several intensity functions obtained from a representative cat. The cochlear response is given in microvolts and the intensity of the stimulus is given in decibels (85 db of attenuation equals 1 dyne per sq cm). The function obtained prior to the application of cocaine (Control, right ear) shows the typical relationship between the intensity of the stimulus and the magnitude of the cochlear response. The other functions show the loss in the magnitude of the cochlear response immediately after the withdrawal of cocaine (0 hours) and 2 and 6 hours following withdrawal. For a stimulus whose intensity was 30 db above 1 dyne per sq cm the normal ear produced a response of about 600 microvolts, whereas the same stimulus produced a response of only 60 microvolts immediately after withdrawal of the cocaine solution. Furthermore, the response continued to drop for a period of 24 hours, at which time it was too small to be measured accurately (less than 0.1 microvolt).

In order to assure that the decline observed was due to the cocaine rather than to a deterioration of the preparation, the other ear was operated upon and the cochlear response recorded. The intensity function for this ear is also plotted in Figure 1 (Control, left ear). Because this function is almost identical to the one obtained 24 hours earlier from the experimental ear prior to the cocaine treatment, it may be concluded that the loss observed in the right ear was due to the effects of the cocaine solution.

*Cyclaine* (5 per cent). The effects of Cyclaine in a 5 per cent solution are similar to those produced by cocaine. Again there is a sudden drop in the response due to shunting. Figure 2 presents intensity functions for a representative animal. A normal function was obtained before treatment (Control, right ear). The other three functions show the loss in the response immediately after withdrawal (0 hours) and after 6 and 75 hours. The control function obtained

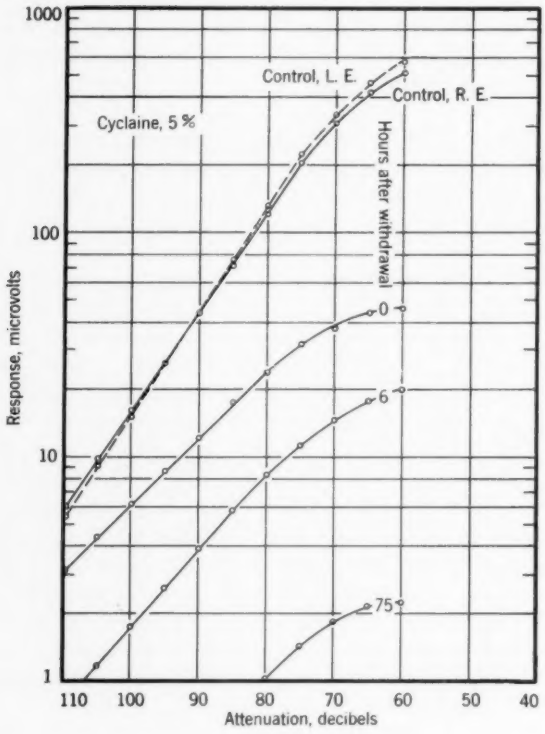


Fig. 2.—Irreversible effects of 5 per cent cyclaine upon cochlear potentials.

on the left ear (Control, left ear) about 96 hours after the right ear was treated with Cyclaine indicates that the loss observed in the right ear was due to the effects of the Cyclaine and not to a deterioration of the preparation.

*Cyclaine* (1 per cent). The effects of the 1 per cent solution of Cyclaine were considerably different from those produced by either cocaine or the 5 per cent solution of Cyclaine. Figure 3 presents intensity functions of a representative animal. Recovery is demonstrated by an increase in output at 3, 8, and 20 hours following withdrawal. At the end of the recovery period of 20 hours the response was almost identical to the control values prior to the application of the anesthetic solution.

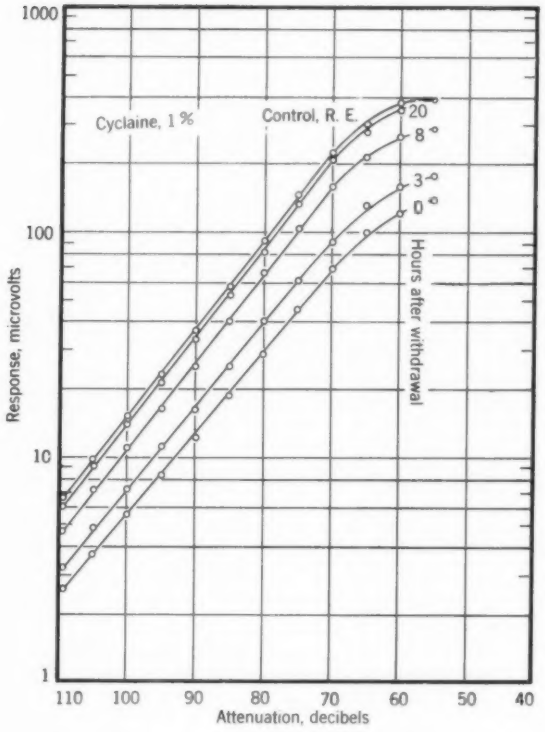


Fig. 3.—Reversible effects of 1 per cent cyclaine upon cochlear potentials.

## COMMENT

Because of the shunting effect observed whenever any of the topical anesthetics or physiological saline was applied to the round window membrane, it was impossible to determine the direct effects of these solutions upon the magnitude of the cochlear response *during* the period of application. However, once the solutions were withdrawn and the electrode field cleared, the magnitude of the response reflected the effects of the anesthetic substances without their obscuration by the shunting.

It is known that permanent damage to tissues may result from improperly prepared solutions of anesthetic agents. Aqueous solutions

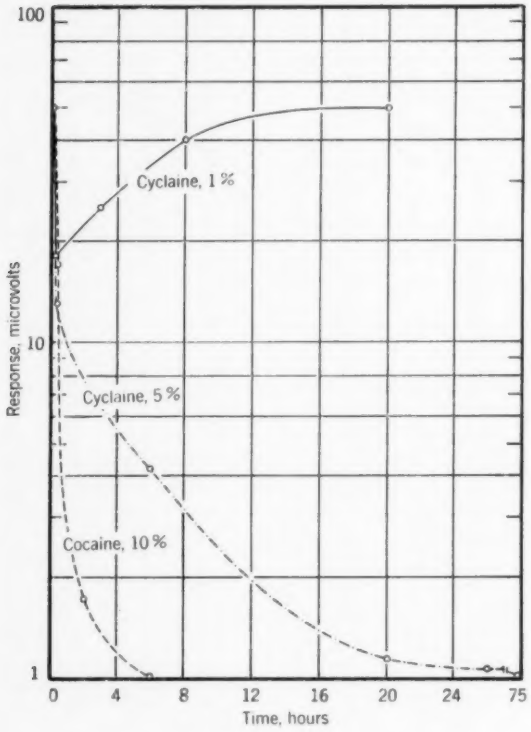


Fig. 4.—Simultaneous comparison of 10 per cent cocaine, 5 per cent cyclaine, and 1 per cent cyclaine with respect to time.

of salts of local anesthetic drugs may exert an osmotic pressure greater or less than that of tissues depending upon their strengths. Hypertonic or hypotonic solutions disturb the osmotic equilibrium between the cells and their external environment.[2] This study made use of properly prepared isotonic solutions; therefore our results are not vitiated by disturbances in osmotic pressure when the anesthetics are applied.

The effects upon the cochlear response of 1 and 5 per cent Cyclaine and 10 per cent cocaine are shown in Figure 4. Of these three solutions, only the application of 1 per cent Cyclaine was fol-

lowed by recovery. During the period of application the response dropped from 50 to 18 microvolts. Within 8 hours of withdrawal the response had risen to 40 microvolts and complete recovery was noted after 20 hours.

The effects of 5 per cent Cyclaine and 10 per cent cocaine were especially damaging, and although the rate of decline in the response was different for these two substances, in all instances the response was ultimately abolished. The brief application of cocaine in meager quantity was sufficient to eliminate the cochlear response within 6 hours. No recovery was observed for periods as long as 60 hours.

The losses produced by the 5 per cent Cyclaine were similar to those just described for cocaine, although the rate of decline was slower in the case of Cyclaine. No recovery was noted for periods up to 90 hours, at which time the cochlear response was completely gone.

Based upon data from this experiment it seems clear that neither the 10 per cent cocaine nor the 5 per cent Cyclaine solution is a suitable anesthetic substance for middle ear surgery. This is especially so since common practices involve doses far in excess of the 0.25 cc used in the present experiment. Moreover, periods of application are apt to be longer than 15 minutes. Solutions of 1 per cent Cyclaine seem to produce no irreversible damage to the hair cells.

The manner in which these anesthetics affect the cochlear response is unknown. In applying these substances to the round window membrane, care was taken not to let the solutions come in contact with any middle ear structures besides the round window. It seems likely, therefore, that the changes represent a direct effect on the hair cells of the organ of Corti.

109 Eno Hall, Princeton University

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LXXIV

# THE ENDOSCOPIC MANAGEMENT OF CONGENITAL ATRESIA OF THE ESOPHAGUS WITH OR WITHOUT TRACHEO-ESOPHAGEAL FISTULA AND OF CONGENITAL TRACHEO-ESOPHAGEAL FISTULA WITHOUT ATRESIA OF THE ESOPHAGUS

BLAIR FEARON, M.D.

TORONTO, CANADA

The problem of congenital atresia of the esophagus with or without tracheo-esophageal fistula and of congenital tracheo-esophageal fistula is not ordinarly considered to be within the province of otolaryngology but rather with in the field of thoracic surgery. However in this hospital, and especially in recent years, the management of this formidable problem has involved the departments of surgery, pediatrics, anesthesia, radiology, and as well, otolaryngology.

Congenital atresia of the esophagus was first recognized and reported by William Durston[1] in 1760. His case has been erroneously referred to by many writers as one of esophageal atresia with a tracheo-esophageal fistula, but Haight[2] has clarified this mistaken impression. The first description of a case with the common anomaly, esophageal atresia with a tracheo-esophageal fistula, was first reported by Thomas Gibson[3] in 1697. Lamb[4] in 1873 was the first to report a case of tracheo-esophageal fistula without other esophageal anomalies.

Many attempts were made to correct these anomalies[5-8] but it was not until 1939 that a successful operation was performed. With only a day between their operations and working independently, Ladd[10] and Leven[11] each were successful in repairing the defects in patients with esophageal atresia with tracheo-esophageal fistula. In the same year Imperatori[12] reported a successful closure of a tracheo-esophageal fistula by a tracheotomy approach. Since these first case reports,

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From the Department of Otolaryngology, the University of Toronto and the Hospital for Sick Children.

Presented as a candidate's thesis to the American Laryngological, Rhinological, and Otological Society, Inc.

surgery for the correction of these anomalies has steadily improved, so that since 1950 the mortality rate for the period of one year has been reported to be as low as 11.1 per cent by DeBoer and Potts.[13] At present, unless there are other associated anomalies incompatible with life, or gross prematurity, and unless the diagnosis has been unduly delayed, the prognosis in such patients is relatively good.

In reviewing such reports one is impressed by the insignificant attention afforded to the role of bronchoscopy and esophagoscopy in the diagnosis and management of such patients either in the pre-operative or postoperative stages. In their textbook "The Esophagus," published in 1958, Benedict and Nardi[14] stated, "Endoscopy has not been utilized as a diagnostic aid in these cases."

It is the purpose of this paper, based on a review of 201 cases admitted to this hospital and on the experience of the department of otolaryngology with many of them, to emphasize the importance of the part the otolaryngologist may play in the endoscopic management of patients with atresia of the esophagus, atresia of the esophagus with tracheo-esophageal fistula, and tracheo-esophageal fistula without atresia (the so-salled H. type of tracheo-esophageal fistula).

## TYPES OF ANOMALY

Vogt[15] in 1929 was the first to classify congenital atresia of the esophagus without tracheo-esophageal fistula and with fistula. He apparently was not aware of tracheo-esophageal fistula without esophgeal atresia for such is not in the classification.

The most common anomaly in this series was that in which there was an atresia of the esophagus and a fistula between the trachea and the distal segment of the esophagus (Type I). This accounted for 175 of the 201 cases reviewed (87%). The next common anomaly was that in which there was a complete atresia of the esophagus without a fistulous communication with the trachea (Type II). There were 11 cases (5.4%) of this type. There were 8 cases (3.9%) of tracheo-esophageal fistula without atresia of the esophagus (Type III). Type IV, in which fistulas were present between both the upper and lower segments in which there was an atresia of the esophagus, accounted for 4 cases (1.9%). There were 3 cases (1.4%) in which there was an atresia of the esophagus and a fistula between the upper segment and the trachea (Type V).

Other congenital anomalies are commonly associated with the esophageal and tracheal anomalies. Eighty-five of the cases reviewed

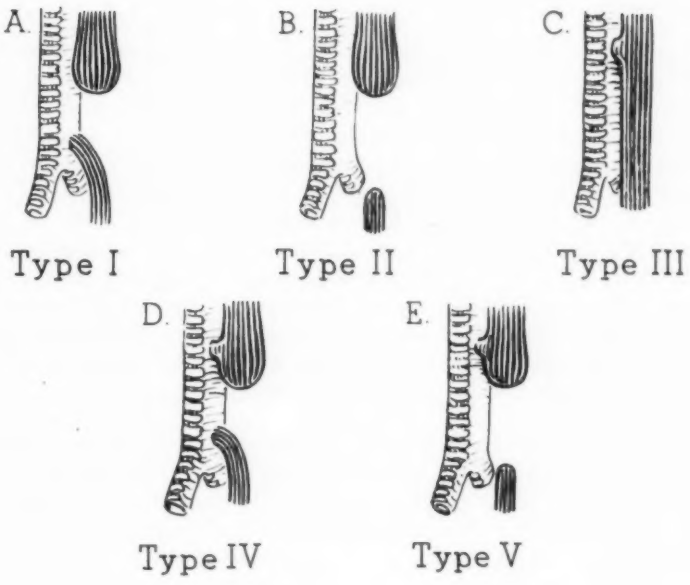


Fig. 1.

had one or more other congenital defects, involving the cardiovascular, neurological, gastro-intestinal, genito-urinary and orthopedic systems. Many of these defects as well required corrective surgery.

## EMBRYOLOGY, ETIOLOGY, AND INCIDENCE

Ferguson[16] and Haight[2] have presented excellent detailed descriptions of the embryology and etiology of atresia of the esophagus and tracheo-esophageal fistula. Embryologically the foregut gives rise to the larynx, trachea, lungs, esophagus and stomach. The trachea and esophagus should have separated and developed normally by the eighth week of embryonic life. The trachea and esophagus appear as a single structure until the third week and this single tube is divided into two by an ingrowth of mesoderm before the twelfth week. It is thought that epithelial lining of the esophagus proliferates, occluding its lumen. It thus becomes a solid tube which is recannulated by a process of vacuolation. If the vacuoles do not coalesce, then an esophageal atresia results. If the ingrowing mesoderm creating the

trachea and esophagus does not completely develop, then a tracheo-esophageal fistula remains.

The etiology of these defects would seem to correspond to the etiology of any congenital defects, rather than a specific cause for esophageal atresia and tracheo-esophageal fistula. Ingalls and Gordon[17] in 1947 suggested possible causes of congenital defects. They considered these in three categories: 1) infections (rubella, syphilis, etc.); 2) chemical (nutritional deficiences, toxic and metabolic disturbances, etc.); 3) physical (mechanical trauma, structural disease of the uterus, etc.). Hydramnios is not an uncommon accompaniment to congenital defects but it is probable that hydramnios in itself may be considered a congenital defect rather than a cause of the congenital defect. Nevertheless, where there has been a hydramnios in the mother, esophageal and tracheal anomalies should be strongly suspected if the newborn infant shows any signs of respiratory distress.

The incidence of congenital esophageal atresia with or without fistula varies remarkably in various centers. Belsey and Donnison[16] have reported a figure as high as 1-800 births and Haight[2] has reported a low of 1-15,432. A reasonable average would appear to be 1-4,000 births.

## EVOLUTION OF ENDOSCOPIC PROCEDURES

In this hospital, primary reconstruction of the esophagus and closure of the fistula for congenital atresia of the esophagus was first attempted in 1944. Subsequently patients with atresia of the esophagus without fistula and tracheo-esophageal fistula were operated also. From January 1944 until July 1958, 201 patients with the above anomalies were admitted to the hospital. The histories of these patients have been reviewed with the object of showing that with the close co-operation of the department of otolaryngology (who perform the endoscopic procedures), the mortality, morbidity and complications have been significantly reduced over the years. It should be pointed out that there has been no selection of patients for surgery; if the patient managed to stay alive long enough to reach the operating room, surgery was attempted. Furthermore, the mortality figures include all patients that did not survive (except one accidental death at home) regardless of their age or cause of death. In short, all the reported recoveries (except the one) are alive and well at the present time.

The author's association with this problem began in 1951 and since then he has bronchoscoped or esophagoscoped, or both, 83 of

TABLE I

COMPARATIVE ANALYSIS OF RESULTS AND PROCEDURES CARRIED OUT IN 201 PATIENTS

1944-1950 and 1951-1958 (First Six Months)

| | |
|---|---|
| Total number of patients from January 1, 1944 to January 1, 1951 | 53 |
| Number of cases operated | 42 |
| Number of survivals | 8 |
| Number of patients bronchoscoped | 4 |
| Number of patients tracheotomized | 1 |
| Number of patients requiring dilatation (prior to 1951) | 3 |
| Percentage survival rate (operated) | 19.2% |

| | |
|---|---|
| Number of cases from January 1, 1951 to July 1, 1958 | 148 |
| Number of patients operated | 140 |
| Number of survivals | 65 |
| Number of patients bronchoscoped | 103 |
| Number of patients bronchoscoped pre-operatively | 19 |
| Number of patients tracheotomized | 20 |
| Number of patients esophagoscoped | 68 |
| Number of deaths from late complications (1 month - 7½ months) | 8 |
| Percentage survival rate (operated) | 46.4% |

In 1957, 18 patients out of 25, or 72% operated, survived.

these patients. Included are three that were operated on prior to 1951. Sixty-one of the patients are alive and well whereas 22 did not survive because of early or late complications.

In the early stages of the evolution of the surgery for these esophageal and tracheal congenital defects, the department of otolaryngology was called in consultation only when the patient was in extremis from respiratory obstruction or when the esophageal lumen became stenosed after otherwise successful surgery.

Currently bronchoscopy is carried out whenever benefit for the patient is likely to be achieved by the procedure. If pre-operatively there is evidence of gross endobronchial exudate or pulmonary atelectasis, bronchoscopy is performed prior to surgery. It is performed immediately postoperatively when endobronchial secretion has been a problem to the anesthetist during the operation or when the lungs fail to inflate at the termination of surgery.

TABLE II

PRIMARY CAUSE OF DEATH IN 129 PATIENTS

(Post-mortem Examination in 123)

| | |
|---|---|
| Prematurity | 3 |
| Chest (bronchopneumonia, massive atelectasis, pulmonary hemorrhage, aspiration, empyema, etc.) | 70 |
| Cardiovascular system (congenital heart disease, acute dilatation, etc.) | 11 |
| Central nervous system (congenital defects, hemorrhage) | 7 |
| Gastrointestinal system (bowel atresia, perforation, peritonitis, etc.) | 8 |
| Genito-urinary system | 1 |
| Surgical complications (breakdown of anastomosis, esophago-pleural fistula, etc.) | 39 |
| Total | 129 |
| Patients with associated multiple anomalies | 82 |

Tracheotomy is carried out when it appears that one bronchoscopy alone is unlikely to control the tracheobronchial exudate; where the laryngeal airway seems inadequate because of a congenital narrowing or because of edema secondary to resuscitation trauma or prolonged or repeated intubation.

Esophagoscopy is done routinely on all patients that survive, about three weeks after the surgical repair, assuming that the patient is fit for the procedure.

It is noteworthy that in 70 out of 129 patients that did not survive, death was due primarily to chest complications—bronchopneumonia; massive atelectasis from aspiration of food, meconium, amniotic fluid; lung abscess; pneumothorax; empyema; etc. Unfortunately, reflux of gastric secretions via the tracheo-esophageal fistula from the lower esophageal segment tends to cause a chemical bronchitis and pneumonitis which may result in an irreversible pulmonary reaction. Because of these factors, the earlier that these anomalies are diagnosed and definitive treatment carried out, the better the results will be.[19]

## DIAGNOSIS IN CONGENITAL ESOPHAGEAL ANOMALIES

Stephens, Mustard, and Simpson[19] have stated that "the diagnosis of these anomalies is simple if the attending physician thinks of its possibility. The presence of excessive salivation in the newborn, or

evidence of choking during the first feeding, should alert the doctor to the possibility of such an anomaly."

In bronchoscopy of the newborn for diagnosis and treatment of severe respiratory distress, the possibility of tracheo-esophageal fistula should constantly be kept in mind. If a tracheo-esophageal fistula should be present, there is a strong likelihood that during the bronchoscopy the tip of the bronchoscope may enter the fistula and progress into the stomach. If such should occur and not be quickly recognized, an unfortunate ending would result from sudden inflation of the stomach with oxygen, as well as from hypoxia. Although the fistula is commonly within two centimeters of the carina or in the carina itself, it may be found as high as the second tracheal ring in tracheo-esophageal fistula without esophageal atresia. When in the trachea, the fistular orifice is in the posterior tracheal wall although a fistula may be present in one or other main bronchus.

When suspected, the diagnosis of esophageal atresia may be further supported by attempting to pass a duodenal tube into the stomach. If there is an esophageal atresia, the passage of the tube will be obstructed. A false impression may be given by virtue of the tube coiling in the proximal pouch of an atresia or passing into the trachea and on into the stomach via a fistula. (This maneuver is not applicable in the diagnosis of tracheo-esophageal fistula without atresia.) Confirmation of the diagnosis of esophageal atresia may be obtained by the injection of 2 cc of lipiodal by means of a catheter into the upper esophagus and roentgenograms obtained. Ferguson[16] has described a method of diagnosing a fistula where other methods have failed, in which the contrast medium is injected by means of a catheter into the esophagus between two inflated balloons. In our experience to date it has been possible to identify the fistula, in all known cases at least, by bronchoscopy, although a recurrent fistula in one patient presented a difficult problem.

The endoscopic management of congenital atresia of the esophagus with or without a tracheo-esophageal fistula, as well as tracheo-esophageal fistula, is considered first from the standpoint of bronchoscopy, and second from the standpoint of esophagoscopy.

## BRONCHOSCOPY

In a newborn that requires bronchoscopy because of severe respiratory distress, for radiological or clinical signs of atelectasis, because of a history of choking on or apparently aspirating a feeding, the

trachea should always be examined thoroughly for a tracheo-esophageal or broncho-esophageal fistula. In most instances, fistulas are not difficult to find. In fact, if the tip of the bevel of the bronchoscope is in a posterior position in the trachea, it will likely inadvertently enter the fistula. Thus the bronchoscopist may find himself examining the gastric mucosa. As mentioned above, the patient may die before the bronchoscopist becomes orientated. In this series the original diagnosis of tracheo-esophageal fistula by bronchoscopy alone was made in four cases with atresia (Type I), two without atresia (Type III) and one with a recurrence of a fistula.

CASE 1. This six day old premature infant, one of twins, was transferred to this hospital with a diagnosis of hemorrhagic disease of the newborn. She had been vomiting blood and mucus. Although she took feeding eagerly by bottle, she choked and become cyanosed. A duodenal tube was introduced into the esophagus and seemed to proceed into the stomach. Gavage feedings were attempted but were vomited.

On admission to hospital the infant was moribund, with a respiratory rate of three per minute and practically no air entry into the chest. The patient was bronchoscoped immediately and after the bloody fluid was aspirated from the trachea and bronchi, a fistula was visualized in the posterior tracheal wall about one centimeter above the carina. The infant's color improved with oxygen and spontaneous respirations ensued.

The day after admission, surgical repair for an atresia of the esophagus with tracheo-esophageal fistula was performed. However the patient died 36 hours after operation. Post-mortem examination showed a bronchopneumonia, an empyema of the right chest as well as mediastinal abscesses.

It would appear that in this case, when the duodenal tube was first introduced into the esophagus to rule out esophageal atresia, it either coiled up in the upper pouch of the esophagus or passed through the larynx into the trachea, thence through the fistula into the stomach. This case points out the value of bronchoscopy, not only in making a diagnosis, but more especially in reviving a moribund patient so that she could be operated and actually survive for 36 hours.

CASE 2. This patient was admitted two days after birth because of frequent choking spells from birth. An esophageal atresia was ruled out by passing a duodenal tube into the stomach. On admission, the six pound, three ounce baby was in good condition although the respirations were slightly rapid and coarse rales and rhonchi were heard in the chest.

Shortly after admission, bronchoscopy and esophagoscopy were carried out. Bronchoscopy revealed the treachea to be filled with watery fluid. This fluid continued to enter the trachea through a fistula in the posterior tracheal wall at about the level of T-1. Esophagoscopy showed a hiatus hernia and it was felt that the hernia was responsible for the large amount of fluid present in the trachea. (An incidental finding was that the right upper lobe orifice arose from the trachea.)

The following day the tracheo-esophageal fistula was successfully closed and the infant made an uneventful recovery.

The patient was re-admitted six months later with a history of having rather severe coughing spells and repeated bouts of bronchitis since the operation. At this

time laryngoscopy showed the left vocal cord to be paralyzed and bronchoscopy revealed a granulomatous mass at the site of the fistula repair. Unsuccessful attempts were made to pass a small bougie through this area into the esophagus. Previously an esophagogram failed to demonstrate a tracheo-esophageal fistula. Esophagoscopy confirmed the presence of a hiatal hernia. It was concluded that the granuloma was due to an embedded suture and that the choking spells were a complication of the hiatus hernia.

Two years later he was again admitted to hospital. He had developed well although he had periodic attacks of vomiting and coughed frequently on swallowing liquids or solids. Bronchoscopy showed the trachea to be much the same as on the previous examination except that a tiny recess could be seen at the proximal edge of the tracheal granuloma. A No. 4 French radiopaque ureteral bougie was introduced into this recess and it passed on into the esophagus. The tip was brought from the esophagus into the mouth and x-rays taken to locate the position of the fistula. The next day, with the bougie re-introduced into the fistula as above to facilitate surgery a secondary closure was performed with an uneventful postoperative course. Six weeks following this operation, a bronchogram via peroral bronchoscopy was carried out. Aside from the anomalous right upper lobe orifice, the report was that of a normal tracheobronchial tree. Endoscopy showed that the left vocal cord was now functioning normally and although there was still tracheal granuloma, it had lessened in size and was pale rather than inflamed. Biopsy indicated a chronic non-specific granuloma.

In retrospect it is probable that a small fistula had been present from the time of the original repair of the fistula until the secondary closure. The granuloma should have been considered from the first to be a result of a persistent fistula. To date the hiatus hernia apparently has not given sufficient cause to contemplate corrective surgery.

In view of the fact that many of the patients with these anomalies have been fed, the tracheobronchial tree is often found to be flooded with feeding. Furthermore, these patients tend to aspirate secretion from their pharynx continually. Other patients may have aspirated amniotic fluid or meconium at birth. When the pulmonary ventilatio nis obviously impaired by excessive material in the tracheobronchial tree, pre-operative bronchoscopy and aspiration is indicated. Far too often the tracheobronchial tree is affected by reflux of gastric secretion through the fistula in the Type I anomaly and this causes a chemical bronchitis and pneumonitis for which bronchoscopy is of little if any value. Nevertheless, this possibility should not be a deterrent to bronchoscopy.

When the diagnosis has been established and bronchoscopy is indicated, the procedure is carried out immediately before the operation to correct the anomaly and in the same operating theatre. The bronchoscope is passed into the trachea without an anesthetic, the trachea and bronchi are cleared of excess secretion. Then the patient may be anesthetized with an ether-oxygen mixture through the bronchoscope. When the patient is anesthetized, the bronchoscope is withdrawn and replaced by a Magill tube for the remainder of the operation.

### POSTOPERATIVE BRONCHOSCOPY

In several instances, even when pre-operative bronchoscopy has been done, tracheobronchial exudate may be a persistent problem to the anesthetist during the operation, necessitating frequent aspiration of the trachea through the endotracheal tube. If such is the situation, immediate postoperative bronchoscopy may be of value. This is particularly true if the lungs fail to inflate fully or if auscultation indicates poor respiratory function of any part of the lung.

### TRACHEOTOMY

A tracheotomy may mean the difference between life and death in certain poor-risk patients. Tracheotomy is performed on those patients with an impairment of the laryngeal airway from traumatic swelling, e.g., after vigorous attempts at resuscitation, or having been sent to hospital with an indwelling endotracheal tube; from congenital narrowing of the laryngeal lumen; or where the tracheobronchial secretions are likely to be a prolonged problem. Five of the successful cases in the author's series had tracheotomies but on the other hand, there were also five unsuccessful cases that had tracheotomies. Extubating the patients has not been a problem and in those that survived the tracheotomy tube was removed in five to seven days. One must make certain that the tip of the tracheotomy tube does not enter the fistular orifice.

As well as for emergency purposes, it has been our custom to perform bronchoscopy from time to time on those patients that recovered from their initial surgery. In some instances it has been for the investigation of late chest complications; in others the bronchoscopy has been in conjunction with esophagoscopy for dilatation or for routine follow-up.

An unproductive, barking cough is characteristic in these patients long after their recovery without much to find on examination to account for the cough. Unless there is pathology behind it, the cough need not be of concern although apprehensive parents may be difficult to convince on this point. The tracheal cartilages in the patients in which a tracheo-esophageal fistula has been repaired seem to lack the rigidity of the trachea in a normal infant of a similar age and size. This apparent deficiency of the tracheal cartilage seems to be the factor which makes these patients somewhat prone to the distinctive cough, although specific pathology should be ruled out. The cough is much more pronounced during an acute respiratory infection so that the condition is often mistaken for acute laryngotracheitis. To date

no patients have required a tracheotomy under these circumstances. In one patient the left main bronchus remained collapsed for several months although it eventually acquired sufficient rigidity to stay open. In another patient a rather long suture was found attached within the orifice of the former fistula.

Following surgery, the orifice of the previous fistula remains indefinitely as a cul-de-sac of a varying depth in the posterior tracheal wall, in the carina or in a bronchus. It changes little in size or appearance as the child develops. It often has the appearance of a bronchial orifice or it may look like a horizontal slit in the posterior tracheal wall.

If the patient has persistent respiratory symptoms such as repeated bouts of bronchitis or coughing and choking when swallowing, the possibility of a recurrence of the fistula must be considered and investigated.

## TECHNIQUE OF BRONCHOSCOPY

Bronchoscopy, where indicated, may be performed quite safely on any of these patients with esophageal and tracheal anomalies regardless of the age, or the size, or the physical condition of the patient. Even though the infant is seemingly moribund, life can be restored by bronchoscopic aspiration. Important qualifications are however: a) the bronchoscopist must be experienced in the bronchoscopy of the newborn; b) that endoscopes in proper working order and of a suitable size and design be employed; c) that oxygen be used; and d) that aspirators also in proper working order and of a suitable size be on hand.

Since these infants are already in respiratory distress of a varying degree, their tolerance for any additional hypoxia is minimal. Prolonged obstruction to the airway by unskillful attempts to pass a bronchoscope into the trachea may be sufficient to tip the scales against the infant's change of survival. Similarly, introducing the bronchoscope into the esophageal segment or through a fistula into the stomach may end in the same result.

For pre-operative bronchoscopy no anesthetic is used. To expose the larynx we use the Gabrial Tucker newborn anterior commissure laryngoscope. For the bronchoscopy we use the Holinger-Jackson 3 mm x 26 cm, or 3½ mm x 30 cm infant bronchoscope. Once the tip of the bronchoscope has passed into the trachea, the oxygen tube

is connected to the bronchoscope. The tracheal secretions are quickly aspirated. The bronchoscope is advanced in the trachea with the tip of the bevel of the bronchoscope directed anteriorly (the handle up) to prevent the bronchoscope from entering the fistula. If the tip of the bronchoscope is directed posteriorly, it will almost invariably enter a fistula in the posterior tracheal wall. Although the lumens of the right and left main bronchi are easily visualized and the aspirator may be introduced into the lower lobe bronchi, the bronchoscope will not pass much beyond the carina in the newborn. Nevertheless the essential purpose of the procedure can be accomplished. Coughing efforts by the patient will express exudate from the distal bronchi to a point where it can be aspirated.

Endotracheal ether and oxygen is the anesthetic of choice in this center. During pre-operative bronchoscopy, following the aspiration, the induction of the anesthetic may be initiated via the oxygen carrier of the bronchoscope until anesthetic relaxation of the patient has been achieved. The bronchoscope is withdrawn and replaced by a Magill's endotracheal tube for the duration of the operation. Recently many of our cases have been maintained without ether, using $N_2O$, $O_2$, and a relaxant (usually succinyl choline) and controlled respiration.

If bronchoscopy is indicated at the end of the surgical repair (or following the gastrostomy), we prefer to introduce the bronchoscope while the patient is still anesthetized. To lessen instrumental trauma and the possibility of hypoxia we make use of the endotracheal tube as a guide for the introduction of the bronchoscope. With the head in the proper position, the endotracheal tube is visualized through the bronchoscope and followed on its anterior surface until the tip of the bronchoscope is between the tube and the epiglottis. The tube is withdrawn and the bronchoscope advanced simultaneously through the larynx into the trachea. It is now of utmost importance that the tip of the bronchoscope not enter the orifice of the repaired fistula. If it does, the suture line is likely to be torn open with consequent serious, if not fatal, complications. There is no danger of this happening if the tip of the bronchoscope is directed anteriorly.

### ESOPHAGEAL MANAGEMENT

A gastrostomy (usually of the Stamm type) has been performed in the majority of the patients one to three days after the surgical repair of the anomaly of the esophagus. The gastrostomy serves not only as a portal through which the patient may be nourished but also provides a simple access for treatment if a significant stricture should

develop. A few patients that were not gastrostomized have proven to be a much greater problem to treat than those with gastrostomies. Gross[20] has reported that dilatations were necessary in 34 out of 97 in his series, and Swenson[21] has found one-third of his patients have had esophageal dilatations.

Although a nesophagogram with lipiodal is done routinely ten days postoperatively, a true evaluation of the lumen at the anastomosis is not thus provided. Therefore the patient is esophagoscoped (preferably under a general endotracheal anesthetic) at the end of three weeks. The esophageal examination is carried out with a 5 mm x 30 cm standard Jackson esophagoscope. If the lumen is widely patent at the anastomosis, then further treatment is unlikely to be necessary and the gastrostomy tube is removed. The site of the anastomosis is often identified at this time by the presence of sutures in the esophageal wall. These eventually disappear.

If the lumen at the anastomosis is narrowed to the extent that a 4 mm esophagoscope (approximately No. 22 French) will pass only with some resistance, then treatment by dilatation may be necessary, and so stringing of the esophagus is performed. Stringing of the esophagus may be accomplished in one of several ways. If the lumen is adequate, a 3 mm Norris Alnico magnet fitted to a No. 8 French duodenal tube is threaded through the esophagus into the stomach. The latter is inflated by means of the tube and a curved Kelly forcep introduced through the gastrostomy into the stomach will pick up the magnet. A string is attached to the tube and drawn through the esophagus.

If the lumen at the anastomosis is very small then stringing of the esophagus is accomplished safely by retrograde esophagoscopy. A small esophagoscope is introduced into the stomach via the gastrostomy and the stomach inflated. The lower esophageal segment will also inflate and, with or without fluoroscopic guidance, the esophagoscope can be readily advanced into the lower esophagus. A No. 5 French ureteral bougie may be passed quite safely through the esophagoscope into the esophagus and thence into the mouth. A string is tied to the bougie and brought through the esophagus.[22]

Retrograde bouginage with Tucker bougies is the method of choice for dilating the esophageal stenosis. It has the advantage of being perfectly safe, requires no anesthetic, and may be carried out on an out-patient basis. Bouginage is repeated bi-weekly until a No. 22 French size is reached. If the patient is otherwise well at this point,

treatment may be discontinued for six weeks and then re-assessed. If at this time the lumen of the stenosis is not smaller than a No. 22 French, and there has been no feeding problem, it is likely that it is adequate for infant feedings. Otherwise the treatment is continued as long as necessary. No two patients respond in the same manner, so no definite criteria can be laid down. As the child grows, the lumen of the stenosis seems to increase in size, particularly if early treatment is instituted.

Although retrograde bouginage is unlikely to damage the esophagus, precautions should be taken during the procedure to prevent aspiration of pharyngeal secretion or vomitus by keeping the pharynx clear with an aspirator. The treatment must be carried out quickly and gently in the small infant and prolonged compression of the larynx and trachea by the bougies must be avoided.

We have largely abandoned direct bouginage from above, not only because of the greater danger of perforation, but because we feel that this method may increase the amount of fibrosis at the stenosis. Because the mucosa above a stricture is usually redundant, it seems possible that the bougie would roll this redundant mucosa into the stenotic area, traumatizing it and creating further fibrosis.

When the patient who requires dilatation has no gastrostomy, we use Hurst mercury bougies when feasible. Otherwise we have adopted the use of different sized esophagoscopes (or bronchoscopes) as dilators. The lumen is first assessed with a suitably large esophagoscope, e.g., a 5 mm esophagoscope in a fifteen pound child. We then select an instrument that is likely to pass easily and then a larger one. If the latter will not readily pass, we believe that by rotating the tip of the esophagoscope and advancing it slowly but firmly, the band or bands of the stricture will be effaced gradually and without tearing. With practice we have found this to be an effective means of dilatation in many cases.

In two patients in which progress could not be obtained otherwise, a modification of the indwelling bougie technique of Tucker was employed.[23]

We have been rather fortunate in the fact that relatively few complications have occurred from esophagoscopy or dilatation. However, a perforation of the esophagus did result from dilatation by direct bouginage in one patient described below.

CASE 3. This patient had a successful repair of a congenital esophageal atresia with a tracheoesophageal fistula at the age of one day. Before discharge from hos-

pital an esophagogram revealed "a considerable degree of stricture at the anastomosis site." However esophagoscopic examination was not requested at that time.

At the age of 18 months the patient was re-admitted because of an inability to swallow anything but strained foods. Esophagoscopy showed a moderate stenosis equivalent to a No. 10 french. The stenosis was dilated directly with semi-rigid, gum-elastic bougies through the esophagoscope up to No. 15 fr. The patient was well postoperatively until sixteen hours later when he developed severe respiratory collapse. Roentgenograph and physical examination indicated a right pneumothorax. Barium swallow showed an esophagopleural fistula. The chest was aspirated repeatedly, and although a pleural effusion occurred, empyema did not set in. The fistula closed spontaneously by the sixteenth day after the accident and the child was discharged as being well 16 days later. His swallowing was improved following the dilatation in spite of the complication and he has had no further serious difficulty over the past six years.

Because of the complication that happened in this patient, even though the bouginage was done with care, we have abandoned the use of direct bouginage from above with semi-rigid bougies unless the bougie is guided with an indwelling string.

The frequency of dilatation has varied from once a day in an occasional problem case to once after three years in one individual in the author's series. The majority in which treatment was necessary were dilated bi-weekly in the early postoperative period. Except for being a nuisance to the parent, an indwelling string is not a problem. (We tie the two ends of the indwelling string together to make a continuous loop.) On the other hand, the gastrostomy often does present a problem to the parents and unless active treatment is necessary, the gastrostomy tube may be removed and the gastrostomy allowed to close around the string. The string is a safety factor in those patients in which the status of the stenosis is in question. As long as the string remains in the esophagus and out through the gastrostomy, treatment may be readily re-instituted at any time. We have been inclined, especially in the earlier cases, to err on the side of conservatism in discontinuing esophageal treatment. It is preferable in our opinion to do this than have to start all over again treatment by dilatation, even if it occurs only rarely.

Case 4. This two day old, 3 pound 14 ounce baby was transferred to this hospital with an established diagnosis of esophageal atresia with tracheo-esophageal fistula. Surgical repair was successfully executed and in this case a polyethylene tube was passed through the esophagus into the stomach during the operation for feeding purposes. It was removed in twelve days. The patient did well for four weeks when he developed a respiratory infection which was controlled with treatment. Later his temperature fell below 96 degrees and with difficulty was brought back to normal in a few days. For several days he vomited coffee-ground material and then bright blood. An esophagogram showed marked narrowing at the site of the anastomosis.

Fifty-one days after surgical repair and after previous postponement because of the patient's poor condition, esophagoscopy was performed. A severe stenosis was found at the anastomosis and a No. 4 fr. bougie was passed through it with difficulty. A gastrostomy was advised and carried out two days later. Following this

operation the baby developed what appeared to be surgical shock and expired two days later. The post-mortem report indicated prematurity, pulmonary collapse and hemorrhage, and pericardial and pleural effusion.

This case is an example of one in which a routine esophagoscopy three weeks after operation for surgical repair would have detected the stricture formation and steps could have been taken earlier to prevent the serious situation which later developed.

TABLE III

ESOPHAGEAL STATUS IN CASES OF ESOPHAGEAL ATRESIA
(WITH OR WITHOUT A FISTULA)

| | |
|---|---|
| Total number of patients esophagoscoped | 68 |
| Number requiring no treatment | 12 |
| Number dilated from one to six months | 26 |
| Number dilated from six months to one year | 6 |
| Number dilated from one to seven years (six of these were operated prior to 1953) | 12 |
| Number under treatment | 3 |
| Number dilated for stenosis secondary to esophageal hiatal hernia | 3 |

## FOREIGN BODIES

Ingestion of foreign bodies by this type of patient (as with any esophageal stricture) seems to occur much more commonly than in normal children of the same age group. Ten of the 83 patients that we have followed have had to be re-admitted for the removal of esophageal foreign bodies. So far none have had solid endobronchial foreign bodies. Excluding food particles—wads of cotton, coins, a button, a peach stone, pieces of plastic have been removed from the esophagus of these patients. It is interesting that whole cooked peas are a fairly frequent source of trouble in strictures. Probably the parents do not expect that peas are firm enough to block the stenosis.

## ESOPHAGEAL HIATAL HERNIA

Since the incidence of esophageal hiatal hernia in infants and small children is greater than that of atresia of the esophagus, it is not surprising that hiatal hernia should be found in a significant proportion of the patients that have been operated for esophageal atresia. When attempting to anastomose the two segments of the esophagus in an atresia, the surgeon may find it necessary to free the stomach and

bring it partially into the thorax in order to avoid undue tension at the suture line. Esophageal hiatal hernia was demonstrated in 13 of the patients in the series (i.e., 201 patients) and repair of the hernia was necessary in three of them. It is noteworthy also that two of the thirteen had a tracheoesophageal fistula only and one of these was repaired.

CASE 5. This patient had a successful repair for a tracheo-esophageal fistula without atresia at the age of five days. The patient did well postoperatively but was re-admitted at the age of nineteen months because of a history of choking on food, especially solids, for six months prior to admission. Esophagoscopy was performed and a plastic foreign body was found and removed from the distal esophagus. Further examination revealed a rather marked stenosis, admitting only a No. 10 fr. dilator. The esophageal mucosa in the distal esophagus was quite inflamed. The diagnosis was considered to be a stenosis at the esophago-gastric junction, secondary to an esophageal hiatal hernia. This was supported by a subsequent x-ray report.

One month later the hiatal hernia was repaired surgically, followed by an uneventful recovery. Two years later the child had been doing well and esophagoscopy showed the lumen at the esophago-gastric junction to be approximately a 24 fr.

This case history is of interest because of the development of hiatal hernia in a tracheo-esophageal fistula without an atresia of the esophagus.

A negative roentgenogram report does not exclude the diagnosis of esophageal hiatal hernia by any means, and this applies particularly to infants and children. With experience in dealing with the problem, the endoscopist can accurately determine the presence or absence of a hiatal hernia. The method we employ is to pass the esophagoscope down the esophagus until the esophagogastric junction is identified. The tip of the esophagoscope is maintained at the junction until its position is located by fluoroscopy or a roentgenogram. Either of these latter techniques reveals the level of the esophagogastric junction and thus confirms or denies the presence of an esophageal hiatus hernia (or short esophagus). An esophagitis directly above, or a stricture at the esophagogastric junction, is supportive evidence of a hiatal hernia. As is the case in other patients with a hiatal hernia, surgical correction is advisable only when the symptoms cannot be alleviated by conservative therapy. One would conclude that if a patient that has had a successful repair of one of the esophageal anomalies under consideration is not making satisfactory progress with its feedings in spite of an apparently adequate lumen of the esophagus, then the possibility of an esophageal hernia should be considered.

CASE 6. This infant was first admitted to hospital at the age of three days with a history of being blue at birth. This was temporarily relieved by suction but the cyanosis recurred repeatedly. One day prior to admission the child was fed, resulting in choking and cyanosis. Examination on admission showed the patient to be in moderate respiratory distress and rales and rhonchi were ausculated over

both sides of the chest. The abdomen was markedly distended. A feeding tube passed into the esophagus met with obstruction. A lipiodal roentgenogram confirmed the diagnosis of esophageal atresia and tracheo-esophageal fistula. Surgical correction of these anomalies was carried out the day of admission. The following day a gastrostomy and Meckel's diverticulectomy were performed. The postoperative course was not unusual.

Esophagoscopy at the age of three weeks showed the site of the anastomosis to be stenosed to the equivalent of a No. 8 fr. Stringing of the esophagus was done and followed by repeated retrograde dilatations for one month. The child was taking the feeding satisfactorily and was well on discharge. Two weeks later she was readmitted because of dysphagia and was dilated retrogradely for four days from a No. 12 to a No. 18 fr. At the age of one year and after several admissions for esophageal dilatations and for respiratory infections, esophagoscopy revealed a foreign body (a large piece of cotton) at a second stenosis at what was thought, radiologically, to be the junction of the middle and lower thirds of the esophagus. The cause of this stenosis was not apparent by esophagoscopy or by x-ray and it was not until six months later that an esophagoscopic diagnosis of esophageal hiatus hernia was made. Surgical repair of the hiatus hernia was then performed.

Following the correction of the hiatus hernia there was an improvement in the swallowing function of the patient and nine months later the lower stenosis was not apparent by esophagoscopy. However the stenosis at the anastomosis still required treatment and the child was taught to swallow Hurst mercury bougies until a No. 26 fr. bougie could be passed. Seven months later (age 2½ years) a convulsive disorder was diagnosed and treated accordingly. Many further admissions to hospital followed this diagnosis, chiefly for repeated lower respiratory infections as well as for removal of foreign bodies. However, at the age of three years and seven months, esophagoscopy showed the esophageal lumen to be very satisfactory. In fact a 7 mm x 35 cm full-lumen esophagoscope passed into the stomach without difficulty.

The history of this patient is far too long to report in detail but it does illustrate the point that an esophageal hiatal hernia may be present to complicate the problem of the management of a stenosis secondary to esophageal surgery.

Case 7. This patient was first admitted to hospital at the age of six months with a history of choking on feedings since birth. X-rays at the age of one month revealed a tracheo-esophageal fistula. He was admitted to hospital on this occasion because of an acute respiratory infection. He was treated for the latter and taken from hospital by the parents before further investigation could be instituted. Bronchoscopy was first performed at the age of two years. A fistula was cauterized with 35% silver nitrate on four occasions without any apparent change in the symptoms of the patient. In the interval he was treated several times for bronchopneumonia or aspiration pneumonia by bronchoscopy and by medication. The last admission to this hospital was at the age of 13½ years when bronchoscopy showed the tracheo-esophageal fistula "well above the carina and looks to be almost as large as a lead pencil." At this time surgical correction was advised but the parents refused consent. The sequel to this story is at the age of nineteen years, the fistula was successfully closed at another hospital and as far as it is known, the patient is well.

The last patient was not one under the care of the author but has been included in this paper to show that: 1) cautery of the fistula was of no value in closing the fistula; 2) that with medication and bronchoscopic aspirations the patient survived for 19 years without serious consequences. In the interval surgical technique for the

correction of tracheo-esophageal fistulas had advanced to the degree where a good result from such an operation could be expected.

## SUMMARY AND CONCLUSIONS

1) The surgical technique for the correction of atresia of the esophagus with or without tracheo-esophageal fistula and of tracheo-esophageal fistula has made tremendous advances since the first surgical cures in 1939. Today, unless there are serious complicating factors, the patient is likely to survive the operation.

2) Although much has been written about the surgical treatment of these anomalies, except for a valuable paper by Ferguson,[16] there has been only casual reference to the role of endoscopic diagnosis and of the management of the complications.

3) The embryology, etiology and incidence are discussed briefly.

4) The evolution of the endoscopic participation of the department of otolaryngology in this center, from being consulted only in an occasional emergency situation to being consulted in each serious respiratory problem as well as carrying out routine esophagoscopy at a certain stage, is described.

5) Figures showing a comparison between endoscopic procedures and surgical results in the periods from 1944 to 1951 and from 1951 to July 1958 are presented. Also included is a table showing the primary cause of death in 129 patients in the series.

6) The history, the passing of a duodenal tube into the esophagus and a roentgenogram with a contrast medium, establishes the diagnosis in most cases. On the other hand the bronchoscopist may make the diagnosis where it had previously been unsuspected.

7) In addition to its application in diagnosis, bronchoscopy has proven to be of distinct value before, as well as after operation, to aspirate excess secretions from the tracheobronchial tree and thus improve the pulmonary ventilation. Tracheotomy is indicated particularly in poor risk patients when tracheobronchial exudate is likely to be a prolonged problem or when the laryngeal airway is impaired. Later diagnostic bronchoscopy is employed in the investigation of persistent pulmonary symptomatology.

8) The technique of bronchoscopy applicable to the problem of performing the procedure safely in a tiny patient with respiratory distress is outlined. The danger of introducing the tip of the broncho-

scope into a tracheo-esophageal fistula either before or after operation must be avoided.

9) At the end of three weeks, routine esophagoscopy (unless contraindicated medically) is carried out to assess the status of the esophageal lumen. When significant stenosis is detected, stringing of the esophagus is executed for the purpose of retrograde dilatation. Dilatation is continued until the esophageal lumen is judged to be adequate to enable the patient to take a normal feeding. The techniques used in these procedures are described.

10) The ingestion of foreign bodies seems to be more prevalent in these patients than in the general population.

11) Esophageal hiatal hernia has been discovered in several patients in the series. This defect may be due in part to the surgical correction of the esophageal anomaly but also has been found in patients with a tracheo-esophageal fistula only. The possibility of esophageal hiatal hernia should be suspected if the patient is dysphagic in spite of an adequate esophageal lumen at the anastomosis.

12) Case histories have been presented to illustrate particular problems related to pre-operative and postoperative endoscopic management of patients with congenital esophageal or tracheal anomalies, or both.

13) Eighty-three of the total of 201 patients have been under the care of the author for endoscopic procedures. From this experience it is the author's conclusion that bronchoscopy, on occasion, can be a life-saving measure and in many patients it undoubtedly reduces the morbidity and postoperative complications. Consequently the duration of the convalescence may be lessened appreciably. Furthermore it is the author's opinion that difficult postoperative esophageal problems may be anticipated by routine esophagoscopy about three weeks following surgical repair. In experienced hands there should be no danger to the patient by these endoscopic procedures.

14) In the final analysis, each patient is an individual problem in management and so a fixed routine for bronchoscopy, tracheotomy, esophagoscopy, or dilatation cannot be applied to all patients.

308 Medical Arts Bldg.

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LXXV

# EXPERIMENTAL FRACTURE OF THE STAPES IN CATS

GEORGE T. SINGLETON, M.D.

AND

HAROLD F. SCHUKNECHT, M.D.

DETROIT, MICH.

In April of 1952, the stapes mobilization operation was reborn in the hands of Samuel Rosen.[8] Since then, increasing numbers of surgical procedures have been performed on and about the oval window. With this increased surgical activity, it becomes increasingly important to understand the reaction of the stapes, incudostapedial joint, stapediovestibular joint and membranous labyrinth to mechanical injuries in the region of the oval window.

In a recent experiment, Bellucci and Wolff,[4] working with cats and monkeys, found that experimental fractures of the stapes were frequently healed by bony union with little or no damage to the labyrinth. On the other hand, Altmann and Basek,[1] in a similar study on rabbits, found infrequent bony union and frequent labyrinthine damage. The explanation for the differences in findings in these two studies is not readily apparent unless it is due to special related factors. Bony union of a fracture of the footplate in the human was reported by Wolff in 1942.[9] There have been several reported examinations of human ears in which fractures of the stapes have been found; but no definite conclusions regarding healing of stapes fractures can be drawn from these reports.[2,5,6,8]

## EXPERIMENTAL PROCEDURE

Healthy adult cats were used in the experiment with only one pre-existing middle ear infection appearing in 14 ears. Anesthesia was

From the Department of Otolaryngology, Henry Ford Hospital, Detroit, Michigan.

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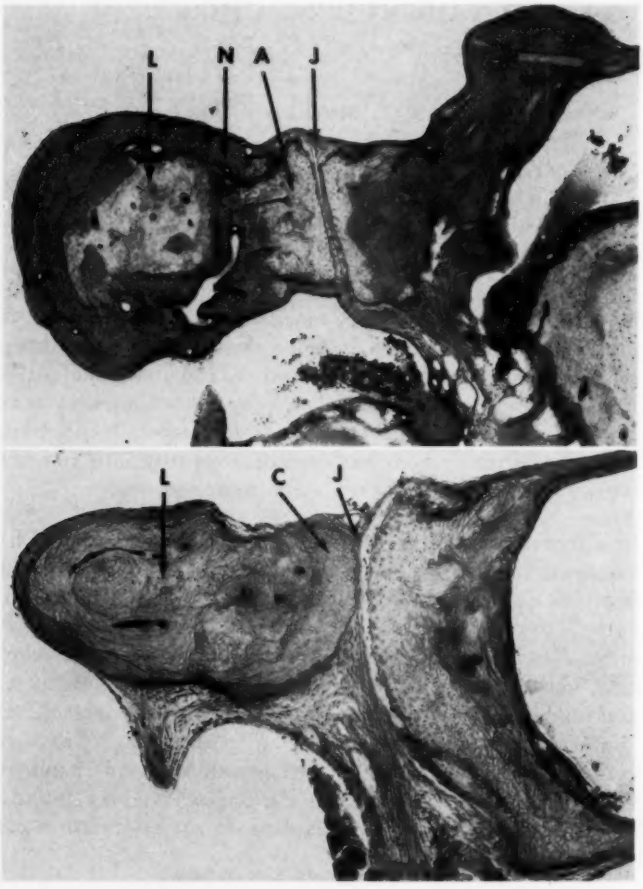


Fig. 1.—*A.* The articular facet (A) which was fractured from the shaft of the lenticular process (L) has reunited by prolific new bone formation (N). The incudostapedial joint (J) is intact. (331 days). *B.* Traumatic separation of the incudostapedial joint (J) with fibrous reunion and degeneration of the cartilaginous facet (C) of the lenticular process (L). (160 days).

accomplished by sodium nembutal injected intraperitoneally. Using aseptic technique, the bulla was opened by an approach posteroinferior to the external auditory canal. The bone was removed to the annulus fibrosis of the drum, care being taken not to injure the tympanic membrane or the skin of the external auditory canal. After removing the bony septum, the oval and round window areas were fully visualized. The mechanical injuries were created with fine needles under high magnification. Procaine penicillin in a dosage of 100,000 units was given intramuscularly once daily for five days. After survival periods of two to eleven months, the animals were sacrificed by intravital profusion with Heidenhain-Susa solution.

The temporal bones were prepared for histological examination by routine methods. The cochleas showing pathology were graphically reconstructed. The following lesions were created:

A. Fractures of the lenticular process—6

B. Separation of the incudostapedial joint—2

C. Fractures of the stapes crura—7 and footplate—7

D. Tears of the annular ligament—6

E. Oval window fistulas—4

F. Diffuse labyrinthine injury—3

G. Lesions of the saccule—8

H. Lesions of the cochlea—4

## RESULTS

A. *Fractures of the Lenticular Process.* Five of six fractures of the lenticular process were produced inadvertently when attempting to separate the incudostapedial joint. In each case, the articular facet was separated from the shaft of the lenticular process. From this, it appears, therefore, that in the cat the fibrous capsule of the incudostapedial joint is stronger than the bone of the shaft of the lenticular process. In five of the six cases, there was complete bony union and, in some cases, the osteogenetic response was prolific (Fig. 1A).

B. *Separation of the Incudostapedial Joint.* The capsule of the incudostapedial joint was torn in two ears. In both of these, the joint space was obliterated by dense fibrous connective tissue. This connective tissue appeared to hold the lenticular process in firm approximation to the head of the stapes. Although this pathological change

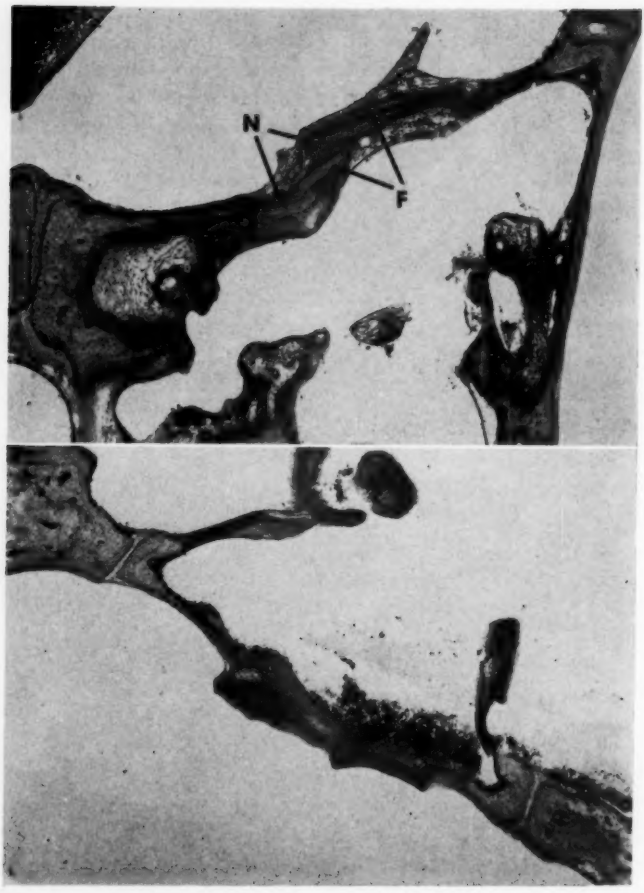


Fig. 2.—*A.* Comminuted fracture of the anterior crus with near approximation of the fragments (F). Complete reunion by new bone (N). The bony fragments of the anterior crus are devitalized as shown by absence of osteocytes. (324 days). *B.* Complete bony union of a comminuted fracture of the footplate of the stapes. A group of chronic inflammatory cells is seen in the intercrural space. (240 days).

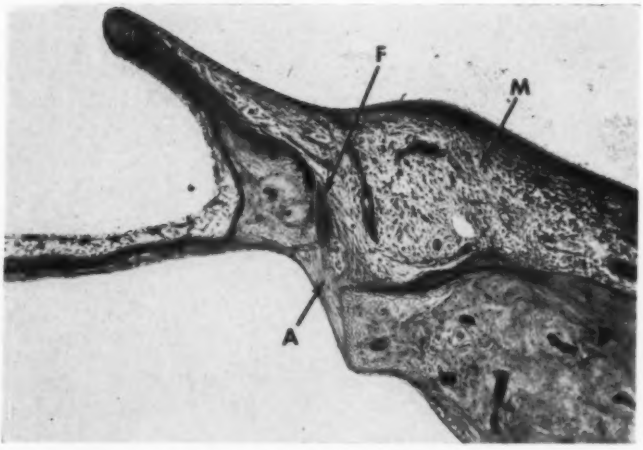


Fig. 3.—Outward subluxation of the anterior margin of the footplate (F) with fibrous healing of the annular ligament (A). The adjacent mucosal lining (M) is moderately thickened. (230 days).

obviously impaired the sliding motion of the joint, it seems doubtful that auditory acuity would be significantly altered. In one of these, there was moderately severe degeneration and resorption of the cartilage of the articular facet of the incus (Fig. 1B).

C. *Fractures of the Crura and Footplate.* Osteogenetic healing appears to occur equally well in fractures of the crura and footplate.

Firm bony healing occurred in five of seven crural fractures. Even in cases with extensive comminution of bone, there was strong bony union (Fig. 2A). Of the two which failed to unite, there was wide separation of the fragments in one and in the other there was beginning osteogenesis in connective tissue which was firmly binding the fragments.

Bony union was complete in four of seven fractures of the footplate (Fig. 2B). One non-union occurred in a stapes which fell from the oval window nine days after mobilization to lie free in the tympanic cavity and in this case, there was bony resorption without healing of the footplate fenestra two hundred days after operation. Of the remaining two non-unions, one was bridged by a muco-endosteal membrane but in the other fistula remained two hundred ninety days after surgery.

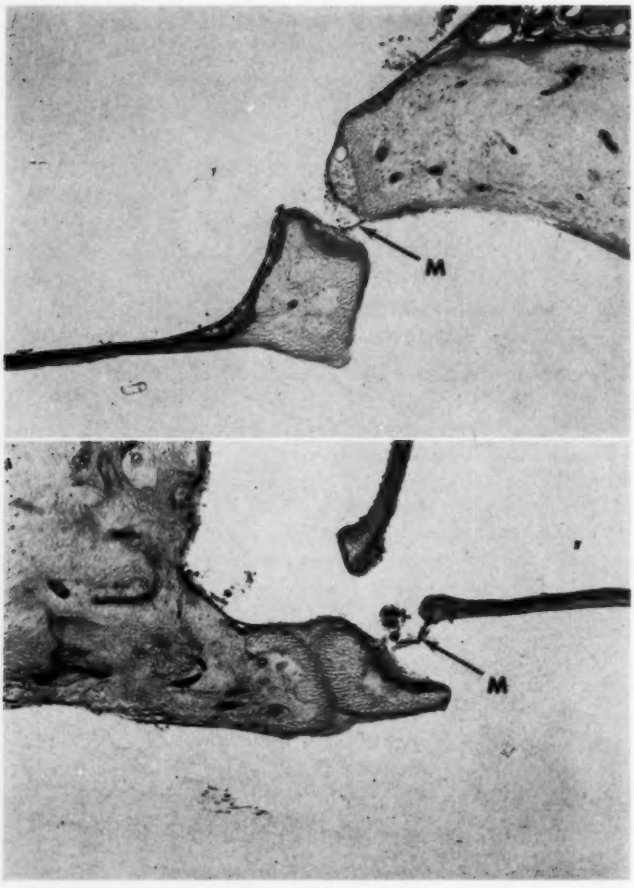


Fig. 4.—*A.* Inward subluxation of the stapedial footplate. The gap at the stapediovestibular joint has been closed with a thin muco-endosteal membrane (M). (260 days). *B.* Fracture of the stapedial footplate with separation of bony fragments and closure of the fistulous opening with a thin muco-endosteal membrane (M). (160 days).

An interesting phenomenon was the laying down of new periosteal and endosteal bone in areas which were near but not at the site of a fracture. The original bone in these areas was devoid of osteocytes, the implication seeming to be that devitalization of the bone, possibly through loss of blood supply, had stimulated new bone growth.

D. *Tears of the Annular Ligament.* In six ears, the annular ligament was torn by subluxation of the stapes footplate or by complete luxation and replacement of the stapes. In five of these cases, the stapes healed back into the oval window margin, the other case being the one in which the stapes apparently fell out of the window as previously stated. In all instances where the replacement of the stapes footplate resulted in close approximation, there was healing of the annular ligament tear with firm connective tissue (Fig. 3). In none of the stapes subluxations was there evidence of osteogenesis at the margins of the oval window or footplate, the reason probably being that the cartilaginous articular surfaces are not capable of supporting an osteogenetic reaction.

E. *Oval Window Fistulas.* When a gap existed between the footplate and the margin of the oval window so that there was separation of the torn margins of the annular ligament, the healing process took place in a different manner. This also applied to fractures of the footplate where approximation was not good. In both cases, neither fibrous nor bony union occurred but a thin membrane was formed to seal the defect. The membrane consisted of an outer or tympanic layer of mucosa one cell layer thick and an inner layer of endosteum which was also one layer thick with no apparent intervening fibrous tissue. That such a thin membrane could grow across the gap, indicates that the perilymphatic pressures were low (Fig. 4, A and B).

In one animal, a patent fenestra of the footplate persisted 290 days after surgery in spite of bony healing of the adjacent fractured crura. This fistula may have remained open because of the magnitude of the bony gap in the footplate. In this ear, there was a moderately severe diffuse aseptic labyrinthitis. There was a group of polymorphonuclear leucocytes extending from the intercrural space through the fistula into the vestibule suggesting an impending suppurative labyrinthitis (Fig. 5).

That an open fistula in the footplate could exist 290 days without profound damage to the sense organs suggests the possibility that cases of sudden deafness or slowly progressive hearing loss following stapes

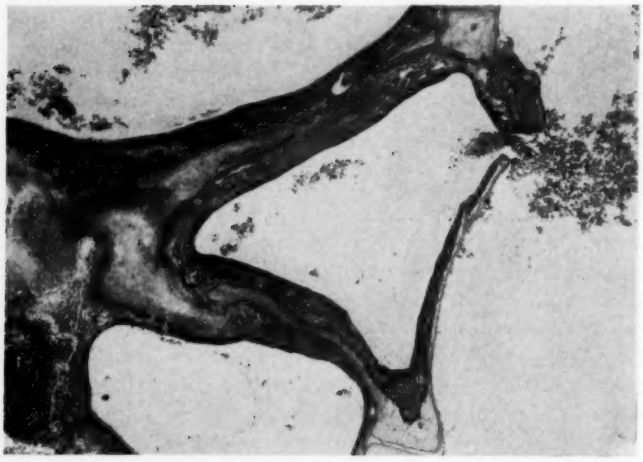

Fig. 5.—Persistent fistula (F) 290 days after comminuted fracture of the footplate. Other fractures of this stapes were healed with new bone. A mild early serious labyrinthitis existed although the animals showed no signs or symptoms of vestibular deficit. A strand of leucocytes extended from the intercrural space through the fistula into the vestibule suggesting an impending suppurative labyrinthitis.

surgery or following temporal bone fracture could be due to persistent oval window fistulae. The dangers of such fistulae are quite obvious for in such an ear otitis media might well result in labyrinthitis and meningitis. It would appear that in performing oval window surgery care should be used to avoid creating and leaving fistulous openings in the oval window area.

F. *Diffuse Labyrinthine Injury*. In two ears, operation resulted in degeneration of the membranous labyrinth and connective tissue proliferation in the inner ear spaces. One was from inadvertent injury to the horizontal semicircular canal and the other from apparent extrusion of the stapes from the oval window on the ninth postoperative day.

The operative procedure in the latter case consisted of completely removing the stapes from the oval window and immediately replacing it. The repositioning appeared excellent and there was no evidence of leakage of perilymphatic fluid. On the ninth postoperative day, the animal first demonstrated head tilt, severe nystagmus and vomit-

ing. The symptoms of vestibular destruction were obvious during the subsequent seven months. Histological examination revealed the stapes to be tilted backward and it could be postulated that the stapedius muscle may have pulled the stapes out of the window in response to a strong auditory stimulus. It is also possible that the stapes luxated much earlier and that a fibrin clot sealed the oval window during the first days after surgery only to give way later and result in diffuse labyrinthitis.

G. *Lesions of the Saccule.* Lesions of the saccule were created in all eight ears in which footplate and the annular ligament fenestrations were made. Of the eight cases of saccular damage, three were associated with diffuse labyrinthine changes as already described. The site of injury to the saccule was always located directly beneath the footplate of the stapes. In one ear, the fenestration needle appeared to have made a discreet lesion in the saccular macula (Figs. 6, A and B). Anson and Bast[3] have recently pointed out the close approximation of the saccule to the anterosuperior margin of the stapes footplate in the human.

H. *Lesions of the Cochlea.* There was rupture of Reissner's membrane and injury to the organ of Corti in the upper basal turn of four ears. In each of these four ears, there was an associated injury to the saccular macula. In only one ear did a lesion of the organ of Corti fail to occur when a saccular lesion was present. It was surprising to us to find that the spacial distribution of the lesions of the organ of Corti which resulted from these oval window punctures was like that resulting from high intensity noise and from head blows. The lesions were less discreet, however, than stimulation injuries.

The histological changes in the cochlea were very similar to stimulation injuries; that is, damage most severe to the outer hair cells and less severe to the supporting cells and inner hair cells (Fig. 7). In all four ears, the lesions of the organ of Corti were associated with tears in Reissner's membranes and injury to the saccule. It may be that compression of the saccule by either an instrument or by an inward displacement of the footplate of the stapes results in a transmission of endolymphatic fluid from the saccule into the cochlea with rupture of Reissner's membrane in the upper basal turn. If this were the case, one would assume that the damage to the organ of Corti was secondary to either the increased endolymphatic pressure or the rupture of Reissner's membrane. McCabe and Lawrence have shown that high intensity acoustic stimulation in the guinea pig results in saccular damage as well as damage to the organ of Corti.[7]

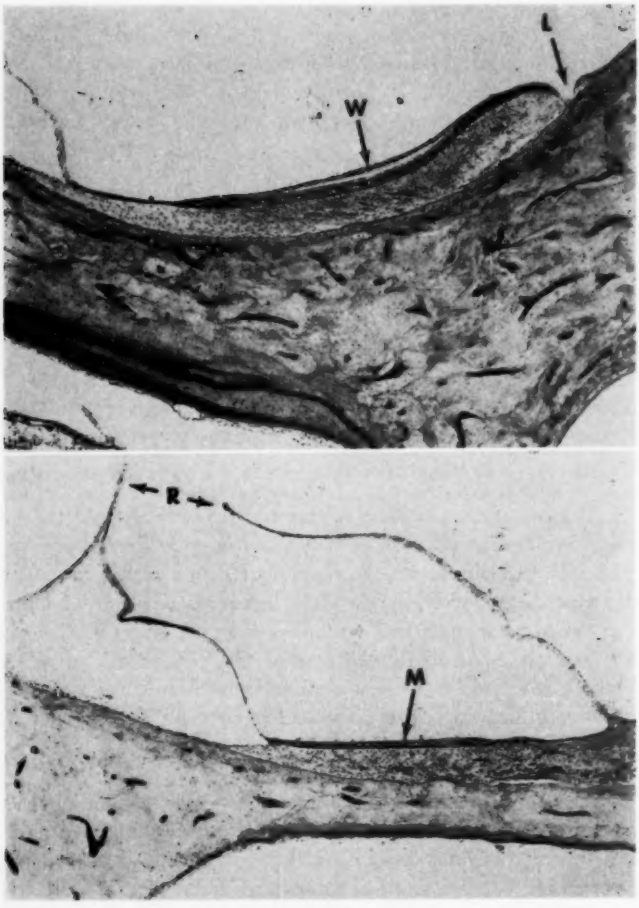


Fig. 6.—*A.* Fenestration of the footplate with a fine needle resulted in rupture and collapse of the saccular wall (W) and a restricted lesion in the saccular macula (L). There was organ of Corti damage in the upper basal and lower middle turns (Fig. 7). The footplate healed by bony union (Fig. 2B). *B.* Inward subluxation of the stapes (Fig. 4A) resulted in rupture (R) of the saccular wall and severe degeneration of the neuroepithelium of the saccular macula (M). (260 days).

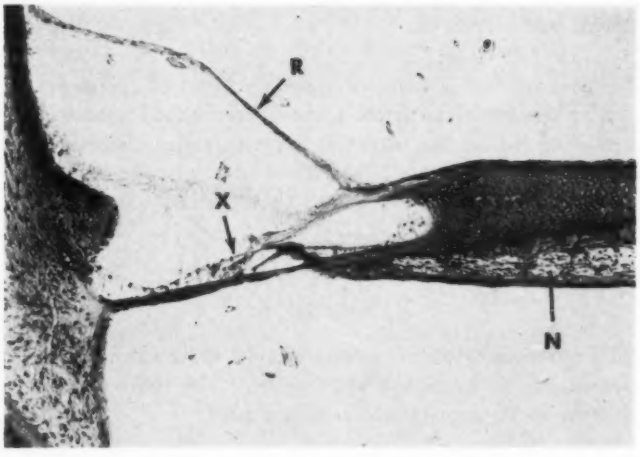


Fig. 7.—*A.* Graphic reconstruction of the organ of Corti showing damage to some of the hair cells in the upper basal and lower middle turns (between 9 and 12 mm). Reissner's membrane in this area had ruptured and healed. There was also partial degeneration of the spiral ganglion in this region. Damage to the saccular wall and macula were present and are shown in Figure 6A. The operation consisted of a needle puncture of the footplate which subsequently healed as shown in Figure 2B (240 days). *B.* Organ of Corti from the 10.1 mm region of the above graphic reconstruction showing a loss of outer hair cells (X), partial collapse of Reissner's membrane (R) and degeneration of nerve fibers (N). (240 days).

SUMMARY

Mechanical injuries were produced about the oval window structures in the ears of 14 cats.

Needling of the incudostapedial joint often resulted in fracture of the lenticular process of the incus rather than separation of the incudostapedial joint. Fractures of the lenticular process of the incus healed by an osteogenetic reaction. All fractures of the crura and footplate of the stapes which were in close approximation healed by an active osteogenetic reaction resulting in firm bony union of the fragments. Luxation and subluxation of the footplate of the stapes and reapproximation into the oval window resulted in firm fibrous reunion with no evidence of new bone formation.

Small fistulas occurring in the footplate of the stapes or at the vestibulostapedial articulation were healed by a thin muco-endosteal membrane. A patent fistula persisted in the footplate of one ear for 290 days resulting in only a moderate serous labyrinthitis at the time the animal was sacrificed.

Fenestrating needle injuries to the footplate of the stapes or subluxation of the footplate of the stapes persistently resulted in tearing of the wall of the saccule, degenerative changes in the macula of the saccule, tearing of Reissner's membrane in the upper basal turn and mild diffuse degenerative changes to the organ of Corti in the upper basal turn of the cochlea.

CONCLUSIONS

The question arises as to how closely the results of the animal experiment can be applied to the human. The following statements would seem to be appropriate in this regard:

1. Whereas stapes fractures in cats are healed by bony union, this may be a species related characteristic and further human pathological studies are needed to determine if the healing properties of the human stapes are the same.

2. In the cat, small fistulas created in the footplate or at the vestibulostapedial articulation (as a result of subluxation) often healed with a thin muco-endosteal membrane and it seems reasonable to assume that this might also occur in the human.

3. In the cat, penetrating lesions of the footplate or inward subluxations of the stapes consistently result in injury to the saccule, to Reissner's membrane and to the organ of Corti in the upper basal turn.

Similar anatomical relationships exist in the human ear which suggests that it might also be susceptible to such injury and, indeed, vestibular symptoms and high tone deafness can result from the stapes mobilization operation.

HENRY FORD HOSPITAL

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LXXVI

# THE LATERAL PHARYNGEAL CLEFT

GEORGE R. L. GAUGHRAN, PH.D.

ANN ARBOR, MICHIGAN

There is considerable question concerning the boundaries and relations of the lateral pharyngeal cleft. The purpose of this work is to determine the boundaries of this region, its relations with surrounding spaces, and the existence of any direct communications between the lateral pharyngeal cleft and contiguous spaces. The results are based on the dissection of approximately 35 half heads.

## DESCRIPTION

The lateral pharyngeal cleft is enclosed by a definite and discrete fascial boundary. Medially, it is bounded by the fascia on the lateral surface of the tensor veli palatini muscle, the fascia of the pharynx wall, and the fascia of the styloglossus muscle. Its lateral confine is the medial pterygoid and the interpterygoid fasciae. Anteriorly, above the level of the retromolar trigone, the cleft is closed by the attachment of the fused medial and interpterygoid fasciae to buccinator fascia. Anteriorly, below the level of the retromolar trigone, where the styloglossus is contiguous with the pharynx wall, it is closed by the fascia sweeping lateralward to attach to the mandible at the junction of the body and ramus. A plane of fascia between the pharynx, the styloid process, and the posterior border of the mandibular ramus limits the cleft posteriorly. This potential space is limited above by the base of the skull, below by a fascial bridge between the pharynx and the mandibular ramus. With this general delineation we can consider the boundaries in greater detail.

Covering the superficial surface of the superior constrictor is a fascial sheet, the peripharyngeal fascia (also identified as visceral or buccopharyngeal fascia (Figs. 1, 3, 4, 7 and 8). This sheet affixes

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From the Department of Anatomy, University of Michigan, Ann Arbor, Michigan.

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anteriorly to the edge of the medial pterygoid plate, the hamulus, and along the raphe to the retromolar trigone; here it covers the fibers of the superior constrictor originating from the mandible (mylopharyngeus portion). Below this it fuses to the side of the tongue where it covers the glossopharyngeus fibers of the superior constrictor muscle. Most descriptions of the medial wall do not consider the region above the superior constrictor muscle, yet this area, of the tensor veli palatini muscle, is involved. In this region the peripharyngeal fascia fuses laterally with a fascia which subdivides the cleft.

The ascending palatine artery enters the region medial to the styloglossus muscle, courses close to the pharyngeal wall and, in the upper portion of the cleft, disappears medial to the lower border of the tensor veli palatini muscle. The artery with its venous satellite are contained within a sagittally oriented fascia which I will designate as a vascular fascia. This plane is comparable to the "lame vasculaire" of Truffert[21] or the nerve-vessel sheath of Weintraub.[24] The vascular fascia is a quadrangular membrane attaching anteriorly to the rear border of the medial pterygoid plate, extending from the hamulus to the tubal process or spine (Figs. 1, 3, 4, 7 and 8). Superiorly it adheres to the lower border of the membranous portion and the lateral lip of the cartilaginous portion of the auditory tube reaching from the tubal spine to the anterior border of the tympanic part of the temporal bone. From the tympanic part the attachment of the posterior border is downward along the stylopharyngeal aponeurosis to the point of entrance of the ascending palatine artery. The inferior border of the fascia blends with the peripharyngeal fascia reaching forward from the region of entrance of the aforementioned vessel to the pterygoid hamulus. By virtue of its attachments, the vascular fascia divides the lateral pharyngeal cleft into a medial paratonsillar area and a lateral paramasticatory region. Both these regions contain a variable quantity of fat. The fascia itself may contain fatty tissue, in which case it becomes filmy and indiscrete.

The tensor muscle is covered by a sheet of fascia, the attachment of which outlines a triangular area corresponding to the shape of the muscle (Figs. 1, 2 and 7). The connection of the hypotenuse of the triangle is to the vascular fascia corresponding to a line passing from the region of the spine of the sphenoid to the terminus of the hamulus. There is a thickening in this region of the fascia forming what has been identified by Cazalas[6] as the sphenopharyngeal ligament (Figs. 1 and 7). Superiorly, it attaches to the base of the skull along the lateral border of the scaphoid fossa, then posteriorly along the sphenoid bone, medial to the foramen ovale, to reach as far caudad as the an-

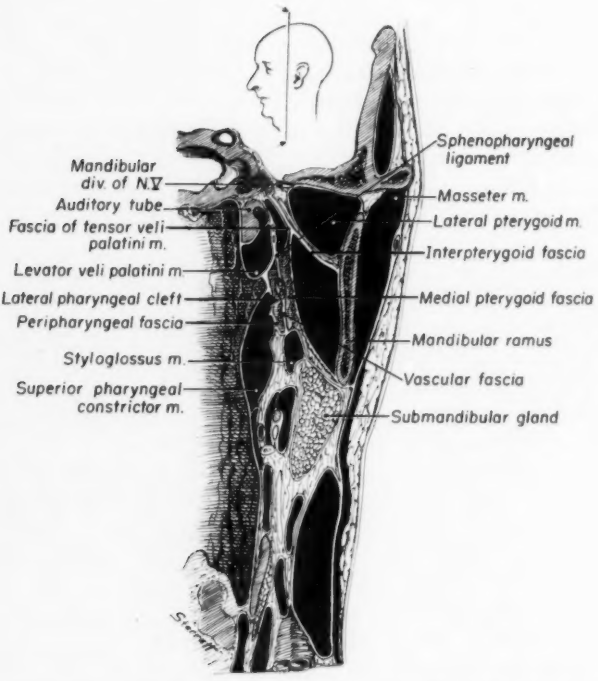


Fig. 1.—A frontal section of the head made at the region shown in the insert and illustrating the boundaries of the lateral pharyngeal cleft.

terior lip of the tympanic part of the temporal bone. Anteriorly it is fastened to the lateral surface of the medial pterygoid plate. This plane was first described by Weber-Liel[23] as a connective tissue membrane of the tensor muscle. Today it is identified by foreign workers as the spheno-pterygo-pharyngeal fascia of Weber-Liel. American workers ignore this plane; I will refer to it as the fascia of the tensor veli palatini muscle.

Though many of us have been unaware of the existence of what has been described here as the vascular fascia, the presence of a fascial plane immediately lateral to the peripharyngeal fascia was pointed out by Escat.[8] He designated this membrane as the "aponeurose lateral du pharynx" and described it as covering the lateral surface of the tensor veli palatini muscle without including the ascending palatine vessels within it. Further, he considered it to be continuous behind

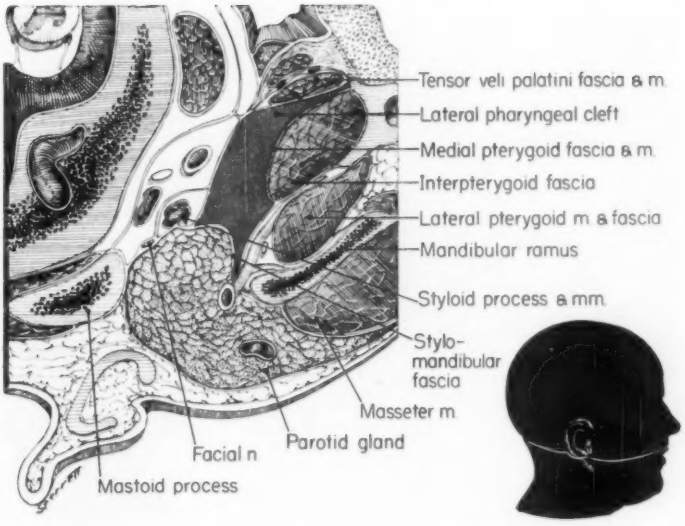


Fig. 2.—A cross-section of the head illustrating the lateral pharyngeal cleft and environs. Level of section shown in insert.

with the "cloison sagittale" which extends from the peripharyngeal fascia at the lateral angle of the pharynx to the prevertebral fascia. The region medial to the lateral aponeurosis he included in the retropharyngeal space. Jonnesco[13] designated the fascia on the lateral surface of the tensor muscle as the lateral aponeurosis of the pharynx and considered it to become continuous with the peripharyngeal fascia at the upper border of the superior constrictor muscle. Soulié[19] and Gérard[9] adhere to the terminology of lateral aponeurosis. These men present a description comparable to that given by Escat. Truffert[20-22] described a "*lame vasculaire*" which is comparable to the plane designated here as vascular fascia. Coulouma[7] and Cazalas[6] presented an analysis of this fascia as part of the study of the spaces adjacent to the pharynx. These workers labelled the region isolated between the lateral aponeurosis and the peripharyngeal fascia the "meta-amygdalen" space and the interval between the lateral aponeurosis and the medial pterygoid muscle the "para-amygdalen" space.

The styloglossus muscle passes downward and medialward in its course to the tongue. In the region of the glossopharyngeus portion of the superior constrictor muscle the styloglossus is flattened against

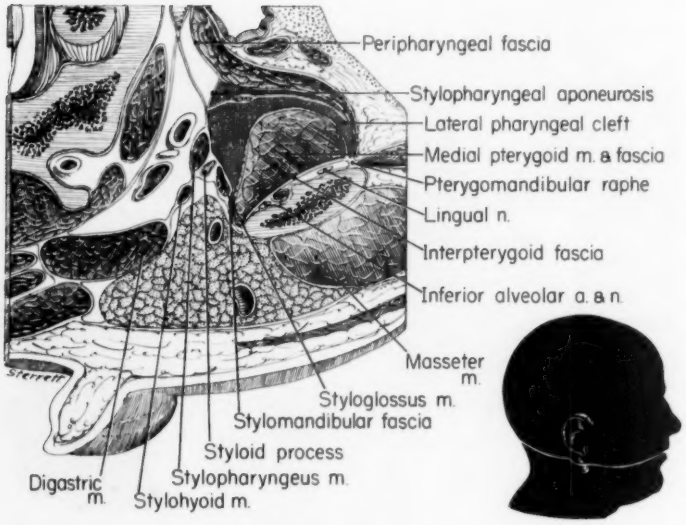


Fig. 3.—A cross-section through the head at the region shown in the insert and illustrating lateral pharyngeal cleft relationships. The cleft is subdivided by a vascular fascia containing the ascending palatine vessels.

the pharynx wall and its fascial covering becomes continuous medially with the peripharyngeal fascia of the glossopharyngeus region and forms the lowermost part of the medial wall of the cleft (Figs. 1, 4 and 7).

Laterally, the medial pterygoid fascia covering the inner surface of the homonymous muscle, forms the major portion of the wall and attaches below to the posterior and ventral borders of the mandibular ramus in the region of the muscle insertion (Figs. 1-8). This connective tissue condensation is generally a loosely woven, delicate plane which cannot be removed intact. In the upper region of the lateral pharyngeal cleft the medial and interpterygoid fasciae fuse with the fascia on the surface of the tensor for a variable distance (Fig. 1). Anteriorly the medial pterygoid fascia is connected to the interpterygoid fascia down to the region of the retromolar trigone and below this point it attaches to the junction of the mandibular ramus and body (Figs. 3, 4 and 5). Behind, along the posterior border of the medial pterygoid muscle, it fuses to the interpterygoid fascia (Figs. 2 and 6). The remainder of the lateral wall, between the posterior

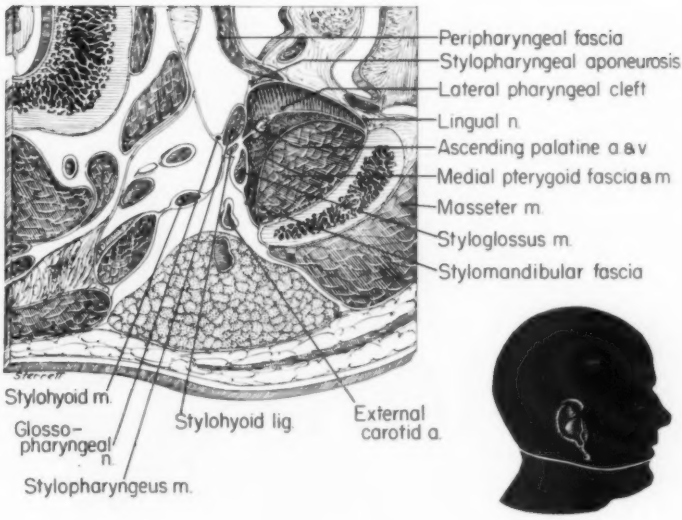


Fig. 4.—A cross-section through the head, at the level shown in the insert, illustrating lateral pharyngeal cleft and its relations. The ascending palatine vessels, within the vascular fascia, are seen as they enter the area.

border of the medial pterygoid and the ramus of the mandible is formed by the interpterygoid fascia. The interpterygoid fascia has superior, inferior, anterior, and posterior connections. Its complete attachment is above, from posterior to anterior: the petrotympanic fissure, the spine of the sphenoid, the sphenoid bone medial to the foramen spinosum and ovale, to the base of the lateral pterygoid plate, posterior and inferior edges of the lateral pterygoid plate to the suture between the palatine and sphenoid bones, thence along this suture to the maxillary tuberosity. Anteriorly, from above downwards, it attaches to the maxillary tuberosity and the buccinator fascia down to the retromolar trigone. Its inferior edge affixes to the inner surface of the mandible: 1) along a line from the retromolar trigone to the lingula, 2) to the free edge of the lingula, and 3) along an oblique line from the lingula to the posterior border of the mandible, above the upper border of the medial pterygoid muscle insertion. The posterior attachment of this sheet connects the first and last points outlined by adhering along the posterior border of the ramus between these two points (Figs. 2, 3 and 5).

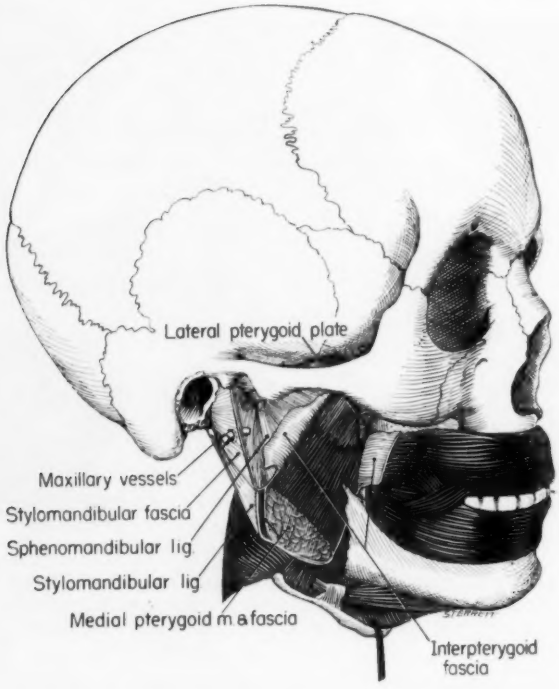


Fig. 5.—Lateral view of the head showing a deep dissection following the removal of masticatory fat pad, mandibular ramus, masseter, temporalis, and lateral pterygoid muscles. Interpterygoid fascia is transected and reflected.

Anteriorly, the lateral pharyngeal cleft narrows and is closed by the attachment of the interpterygoid fascia, to which the medial pterygoid fascia has fused, to the buccinator fascia along the anterior border of the medial pterygoid muscle down to the level of the retromolar trigone (Fig. 3). Below this level the fascia over the styloglossus muscle joins the peripharyngeal fascia and together they sweep lateralward to fuse to the ramus of the mandible at the junction of the ramus with the body (Figs. 1 and 7).

Posteriorly a sheet of fascia stretches from the rear edge of the mandibular ramus to the pharynx wall (Figs. 2-8). Juvara[14] identified this sheet as the stylo-maxillo-pharyngeal aponeurosis. This plane represents a part of what has been described by French workers as

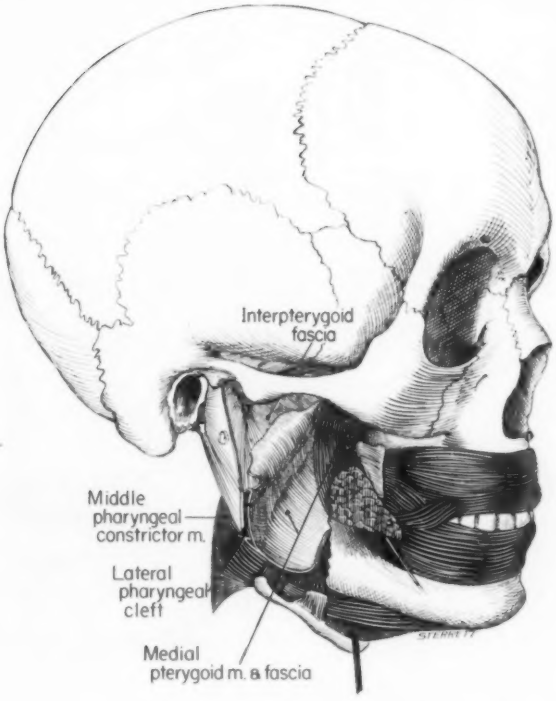


Fig. 6.—Lateral view of the head with the medial pterygoid muscle lapped forward showing a portion of the lateral wall of the lateral pharyngeal cleft. Dotted line represents the line of incision to be made in the medial pterygoid fascia.

the styloid diaphragm (Gilis and Peyron[10]). The more lateral part of the sheet extends from the styloid process and stylopharyngeus muscle to the ramus and can be identified as the stylomandibular fascia (Figs. 3-5 and 8). It attaches above along the tympanic part of the temporal bone from the styloid process to the mandibular ramus. The styloglossus muscle and the stylohyoid ligament are included within the sheet (Figs. 3 and 4). That portion of the stylomandibular fascia adhering to the posterior border of the ramus, above the angle, is thickened to form a fibrous band, the stylomandibular ligament (Figs. 5 and 8).

The more medial portion of the sheet reaches from styloid process and stylopharyngeus muscle to the pharynx wall and was named the

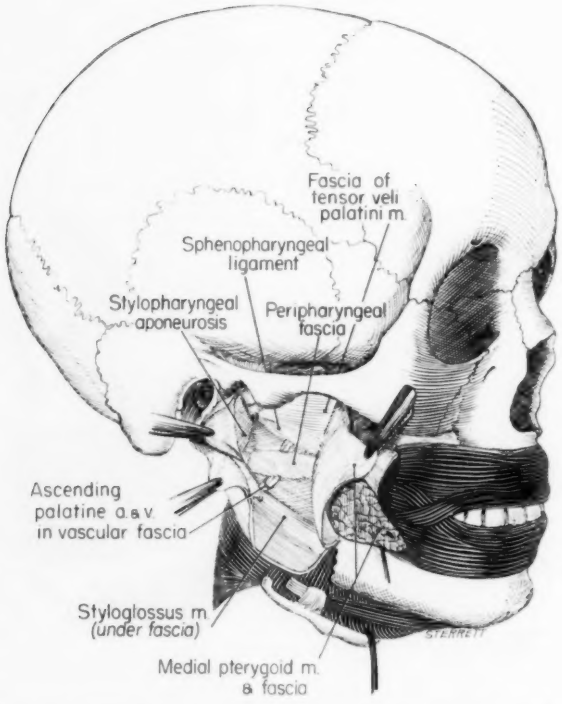


Fig. 7.—Lateral view of the head with the medial pterygoid muscle and fascia lapped forward showing the vascular fascia. A portion of the vascular fascia has been removed to expose the pharyngeal wall covered by peripharyngeal fascia.

stylopharyngeal aponeurosis by Juvara[14] (Figs. 3, 4, 7 and 8). It is also identified as the "aileron." This portion of the sheet fastens above to the base of the skull along the free edge of the tympanic part of the temporal bone extending posterolaterally from the sphenoid spine to the styloid process. The medial attachment to the lateral angle of the pharynx and the lateral attachment to the stylopharyngeus muscle converge inferiorly and meet at the point where the stylopharyngeus muscle enters the pharynx wall.

The upper limit of the lateral pharyngeal cleft is a line on the base of the skull along the sphenoid bone, from the root of the medial pterygoid plate to the sphenoid spine behind. This lies between the

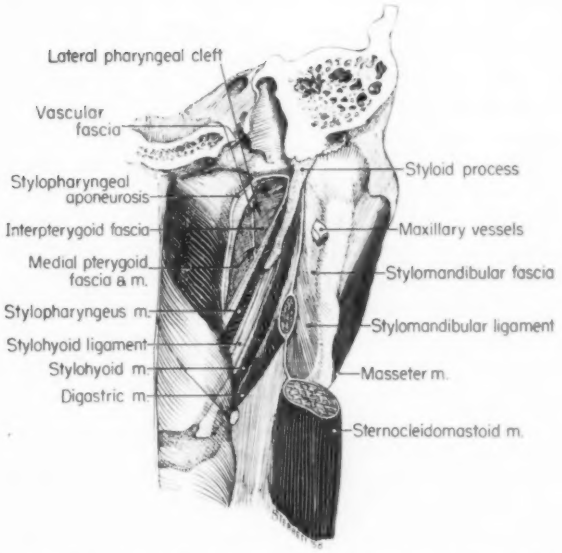


Fig. 8.—Posterior wall of the pharynx showing the stylopharyngeal aponeurosis and stylomandibular fascia which form the posterior wall of lateral pharyngeal cleft. The aponeurosis has been cut and reflected, exposing the cleft. The cut posterior edge of the vascular fascia is visible.

attachment of the medial pterygoid and interpterygoid fasciae laterally and the fascia on the lateral surface of the tensor veli palatini M. medially (Fig. 1). The upper extent of the cleft may not reach the skull because of a fusion of the contiguous fascial planes. Behind this it is limited by the tympanic part of the temporal bone along a line between the stylopharyngeal aponeurosis and the stylomandibular fascia behind and the interpterygoid fascia in front.

Below, the plane of fascia on the anterior surface of the styloglossus muscle attaches to the lower border of the ramus laterally and the peripharyngeal fascia medially thus cutting off the lateral pharyngeal cleft inferiorly (Figs. 1, 4 and 7).

### CONTENTS

In the posterior region of the cleft, the ascending palatine artery enters the area after piercing the fascial sheet between the styloglossus and the stylopharyngeus muscles. With it is the ascending palantine

vein, a tributary of the retromandibular or common facial vein (Figs. 4 and 7). The artery ascends in the vascular fascia and, during its ascent, sends off branches of distribution to the pharynx. This vessel leaves the cleft above to enter the space of the tubal muscles. The tonsillar branch of the facial artery courses on the surface of the superior constrictor muscle but deep to the peripharyngeal fascia, hence should not be included in the cleft. The ascending pharyngeal artery passes superiorly toward the base of the skull, behind the stylopharyngeal aponeurosis and is not to be considered among the contents of the lateral pharyngeal cleft. Numerous small arteries supplying and veins draining the medial pterygoid muscle pass through the fat in the cleft as radicles or tributaries of the ascending palatine artery or vein respectively. Small vessels from the maxillary artery (or from its meningeal branches) may pass medially through this potential space to supply the tensor veli palatini muscle.

In the upper region of the lateral pharyngeal cleft and crossing it obliquely from lateral to medial are the nerves to the medial pterygoid muscle, the nerve to the tensor veli palatini muscle, and the nerve to the tensor tympani muscle. In many cases the fascia on the tensor muscle fuses with the medial pterygoid fascia obliterating any potential space in this region. The glossopharyngeal nerve sends branches around both sides of the stylopharyngeus muscle but these lie medial to the peripharyngeal fascia and are not in the cleft. Some pharyngeal branches, however, may pierce the muscle and these will be found within the cleft. These branches probably also carry fibers of the vagus. Faucial branches of the lingual nerve are given off in front of the anterior border of the medial pterygoid and are not within the lateral pharyngeal cleft. Lymphatic nodes were not seen in gross dissection.

## COMMUNICATIONS

The lateral pharyngeal cleft occupies a unique position among the fascial spaces of the head in that it is directly adjacent to seven other potential spaces. These spaces are: the paralingual space anteriorly, the parotid compartment posterolaterally, the carotid sheath posterocentrally, the retropharyngeal cleft posteromedially, the masticator space laterally, the space of the palatal muscles medially, and the submandibular space inferiorly. Six of these spaces are shown in Figures 1 through 4. However, as described earlier, the walls of the lateral pharyngeal cleft are complete and do not present any direct, open communications with the contiguous spaces. The fascial barrier between the lateral pharyngeal cleft and the submandibular

space is consistently the thinnest region and the stylopharyngeal aponeurosis the densest plane as seen in dissections of cadaver material. When a pharyngeal process of the parotid gland is present, the stylomandibular membrane, which covers the anterior surface of the process and separates it from the lateral pharyngeal cleft, may be very thin and delicate, but, nevertheless, complete.

## COMMENT

The lateral pharyngeal cleft should be thought of as a "cleavage plane" occupied by a loose connective tissue and containing relatively few structures. Equivalents of the term cleavage plane, "Spaltraume" and "Bindgewebsspalten," were used earlier by Henke[12] and König[15] respectively. The term cleft should not be used synonymously with the terms space or compartment. The two latter terms designate a region containing many structures of considerable size; whereas the former designates an interstice, in this instance, between the masticator space laterally and the pharynx wall medially. The lateral pharyngeal cleft is an important entity subject to frequent infection from the adjacent palatine tonsils. A peritonsillar suppuration may erode through the superior pharyngeal constrictor muscle and the peripharyngeal fascia to enter the paratonsillar portion of the lateral pharyngeal cleft. In this circumstance, the plane described earlier as the vascular fascia can play an important role in acting as a temporary or permanent barrier, preventing the lateralward spread of pus to the paramasticatory part of the cleft and adjacent spaces. According to Shapiro,[18] Beck,[2] and Casberg,[5] tonsillar abscess is the most frequent cause of deep neck infection. In fact, a not uncommon sequela of these infections is an erosion of the internal carotid artery with possible severe hemorrhage as cited by several others.

Mosher[16] reported that parotid tumors spread directly to the pharynx wall, and Shapiro[18] reached a similar conclusion based on injections. Barlow[1] and New and Erich,[17] however, stated that injection pressures had to be considerable before an injection mass passed from the parotid compartment to the lateral pharyngeal cleft. On the basis of dissections, this inconsistency between clinical and cadaver data can be seen to be more apparent than real. In all specimens observed by the writer the stylomandibular membrane was complete but the density varied from individual to individual. In some cases the fascia appeared dense and was torn with difficulty; in other specimens the plane was cribriform and was broken with the removal of the pharyngeal process of the parotid gland. Although this fascial plane, the stylomandibular membrane, is present and represents a

complete anatomical barrier between the lateral pharyngeal cleft and the parotid compartment, it cannot always serve as a functional barrier to the extravasation of a purulent fluid from one to the other of these regions. Casberg[5] reported a ready spread of infection from the lateral pharyngeal cleft to the submandibular space. This pattern of extension might be anticipated since: 1) pus will gravitate downward toward the submandibular space and 2) the fascial plane separating this space from the lateral pharyngeal cleft is generally thin. On the basis of cadaver observations, the fascial barriers between the lateral pharyngeal cleft and the carotid sheath, palatal muscles, masticator, retropharyngeal and paralingual spaces are complete without hiatuses. Grodinsky and Holyoke[11] indicated a spread of injection mass from the lateral pharyngeal cleft to the retropharyngeal cleft (space No. 3). This would necessitate a rupture of the stylopharyngeal aponeurosis by either erosion or injection pressure.

I cannot terminate this report without referring to a recent paper by Burman[4] in which it is stated: "The fascial planes have thus been largely man made or artificially made." It cannot be denied that papers on the anatomy of fascial planes and spaces have been colored by the researchers' personal interpretations. In addition, we must agree with a statement cited by Wood[25] that fascial planes do not by any means always form effective barriers against or natural passageways for the course of pus. It is also true that errors have been made. To state that fasciae are largely man made or artificially made, however, is to dismiss a rather knotty and complicated area in which dissection is difficult and in which the literature is surprisingly extensive, rich in varying interpretations and confusing in terminology. To discard this early work is foolhardy. The only intelligent approach to the study of space infections is an examination of clinical data together with an awareness of anatomical structure. These two phases must be brought into harmony before the clinician or the anatomist can clearly understand space infections and reasonably anticipate possible extravasation routes.

## SUMMARY

The lateral pharyngeal cleft is an interstice between the masticator space, the pharynx wall and the tonsillar fossa, the space of the palatal muscles, the paralingual space, the retropharyngeal cleft, the carotid sheath contents, the parotid fossa, the base of the skull, and the submandibular space. It contains the ascending palatine artery and vein in a vascular fascia which subdivides the cleft into paratonsillar and paramasticatory regions. In addition, one finds within the

lateral pharyngeal cleft the medial pterygoid arteries and veins, the nerve to the tensor veli palatini muscle, the nerve to the tensor tympani muscle, the nerve to the medial pterygoid muscle, and a collection of adipose tissue. It occupies a key position relative to the spread of infection from or to any of these contiguous areas, but is separated from these regions by a complete fascial barrier. The fasciae vary in regional density in a fairly consistent pattern in different cadaver specimens and can account for the greater tendency of a fluctuating mass to spread in one direction rather than in another. A knowledge of the spatial relationships between potential spaces and an appreciation of the locations, attachments, and densities of fascial planes allows one to anticipate intelligently the spread of infectious masses and to understand and interpret clinical data on space infections.

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# LXXVII

# ADENOID CYSTIC EPITHELIOMA

## "BROOKE-FORDYCE DISEASE"

FATMA H. ABDIN, M.D.

AND

ABDEL SALAM EL-BARBARI, F.R.C.S.

CAIRO, EGYPT

This disease is described under different names, such as multiple benign cystic epithelioma, tricho-epithelioma, acanthoma-adenoidum cysticum, hydradenoma, benign epithelioma with colloid degeneration, novus follicularis, etc. . . . the best name, however, is adenoid cystic epithelioma or Brooke-Fordyce disease.

In 1891 Brooke[1] described multiple benign growths of the skin under the name "adenoid cystic epithelioma." At about the same time Fordyce[2] was carrying out independently a similar research and published his paper under the title of "Multiple Benign Cystic Epithelioma of the Skin." Their clinical and histological descriptions are practically identical. They differentiated this tumor from other conditions such as tumors of sweat and sebaceous glands. In 1885 Balzer[3] and Menetrier had described clinical and histological pictures quite identical with those of Brooke and Fordyce under the name of sebaceous adenoma of the face and scalp. Perry[4] had also published a beautiful example of this condition under the title "Adenoma of Sweat Glands." Jarisch[5] in 1894 traced the origin of the tumors from the basal cells of hair follicles. In 1922 Stavard[6,7] reviewed the literature and added several other similar cases of multiple as well as solitary tumors.

The disease may appear as multiple nodules or as a solitary tumor.

The multiple growths have certain clinical and histological characters which suggest a congenital origin. The syndrome is commoner in females and has a definite tendency to be familial. This was observed by Brooke, Fordyce and also by Stavard, who reported that

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From the Pathology and Ear, Nose and Throat Departments, Faculty of Medicine, Kasr El-Eini Hospital, Cairo University.

Fig. 1.—Adenoid cystic epithelioma x 30. (Case of multiple growths).

a mother, three daughters and a son of a family of nine were affected and the third child of the eldest daughter was also affected. The disease usually appears at puberty and slowly progresses. Few cases have been reported in children. Goldsmith and Fredenthal[8] saw a case in a boy of seven years, in whom multiple basal cell growths appeared at the age of five. Paul and Inglies[9] saw a child of two years with such multiple familial tumors. Clinically they rarely reach the size of a nut (0.5 to 1.0 cm), are firm painless circumscribed nodules, scattered on the central part of the face, namely, the forehead, eyelids, nose, and cheeks. They may also be seen on the scalp, chest, axilla and limbs but not below the pelvis. They are covered by smooth intact shiny epidermis, and are colorless or slightly yellow, sometimes brown pigment; however, they can be seen under the skin.

The single as well as the multiple adenoid cystic epithelioma has the same histological picture. It consists of epithelial masses, the cells being either basal or squamous, with cyst formation so as to assume a glandular pattern and with keratin masses which look like hair follicles, epithelial pearls or cell nests. Dermal changes may occur around the tumor. The elastic fibers increase in number and size.

They may fuse together and even in H & E sections the elastica may appear a thick (2 to 3 mm) bluish membrane (Willis, 1944).

## REPORT OF CASES

CASE 1. *Example of Multiple Cystic Epithelioma.* L.M.S., a male, aged 25 years, was admitted to the Dermatology Department, Kasr El Eini Hospital, complaining of multiple small nodules of the skin of four years' duration.

Examination revealed a papular lesion of the face and neck, pin head to pea sized, of normal color of the skin and firm in consistency. The diagnosis of syringoma or benign cystic epithelioma was suggested clinically.

Histology of one of the papules showed the picture of benign or cystic epithelioma. The lesion was covered by healthy stratified squamous epithelium with proliferation of the hair follicles forming epithelial masses surrounding spherical red stained keratin material in various stages of degeneration almost to complete disappearance with the formation of cystic spaces (Fig. 1). This picture conformed with the origin from the hair follicles. The cells did not look very active and mitotic figures were uncommon. This was in agreement with the long duration of the lesion. Sebaceous glands were seen attached to and between the hair follicles. The stroma consisted mainly of fibrous tissue and by Verhoff Van Gieson stain, only a few elastic fibers were seen. The lesion was not encapsulated, and appeared as localized abnormal hyperplasia of the hair follicles rather than true neoplasia.

CASE 2. *Example of Solitary Cystic Epithelioma.* N.A.S., a female aged 25 years, was admitted to Kasr El-Eini Hospital, complaining of a mass of the tip of the nose of two months' duration.

The condition started as a small nodule two months ago and gradually increased in size. There was no pain or bleeding and no history of trauma, no relevant personal or family history. Clinically there was a firm small mass arising from the tip of the nose downwards, of the size of a large cherry, slightly lobulated, nodular on the surface. The covering skin was thin, slightly red with fine dilated venules, freely mobile and stretched over the tumor. The mass was deeply fixed to the tissues of the nose, namely, the greater cartilages and their medial crus. The mass was not warm or tender. The nose inside was negative and no glands were present in the neck.

Fig. 2.—Photo of patient with adenoid cystic epithelioma of tip of nose.

An oval incision was made around the base of the tumor. The skin was easily separated and the mass was shelled out from the deeper structures. A piece of the redundant skin was excised and the wound stitched. Gross examination of the specimen showed the mass to have an irregular surface and a firm consistency. It was homogenous and fleshy on cut section with no areas of necrosis.

Microscopically the tumor showed the typical picture of adenoid cystic epithelioma, namely, it consisted of epithelial masses showing glandular arrangement, cyst formation and epithelial pearls (Figs. 3 and 4). In areas it superficially resembled a well-differentiated epithelioma, being formed of polygonal cells, not unlike prickle cells, sometimes differentiating into cells with eleidin brown pigment and into keratin and cell nests. These areas resembled hair follicles. In many fields there was tendency for the cells to be arranged around cavities or cysts, so as to assume a glandular pattern. The lining of the cysts were usually ordinary polygonal cells but sometimes cuboidal or flattened cells, so as to give the impression of being sweat glands or their ducts. These cavities were either empty or contained mucoid material, cellular debris or keratin. The cell masses were separated by pale bluish mucoid material. The cells as a whole appeared very active,

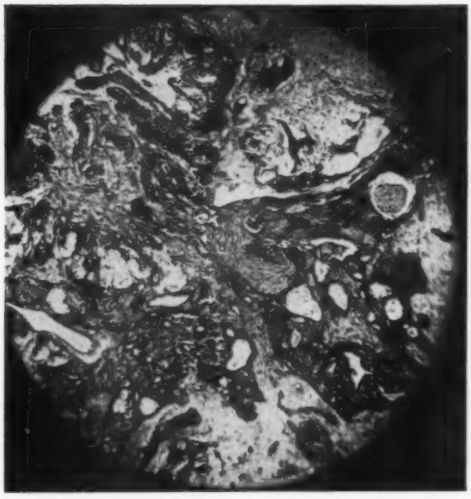

Fig. 3.—Adenoid cystic epithelioma H&E x 2.5. Note the cystic spaces surrounded by epithelial masses.

having large nuclei, either hyperchromatic or vesicular with prominent nucleoli.

## COMMENT

*Origin.* Adenoid cystic epithelioma is best regarded as a type of basal cell carcinoma, which arises from the deep layer of the epidermis or the outer layer of the sheath of the hair follicles. Jarish[5] was the first to trace the origin from the basal cells of the hair follicles. Some observers believe that the tumor arises from sweat glands[10,11] or sebaceous glands.[12] Benign tumors of the skin appendages can easily be differentiated histologically but when they become malignant some cases may be extremely difficult to differentiate from each other. As pointed out by Gates et al,[13-15] such tumors may show the typical structure in only small fields. Another difficulty is that it seems in some cases, the tumor arises from more than one element of the skin appendages. Ewing cited in his textbook on tumors that Dorst and Delbanco observed multiple tumors of the scalp in the same patient, some had the structure of sebaceous adenoma, others of adenoid cystic epithelioma. The tumor resembles also ordinary epithelioma and rodent ulcer. This is not strange however, because all these tumors

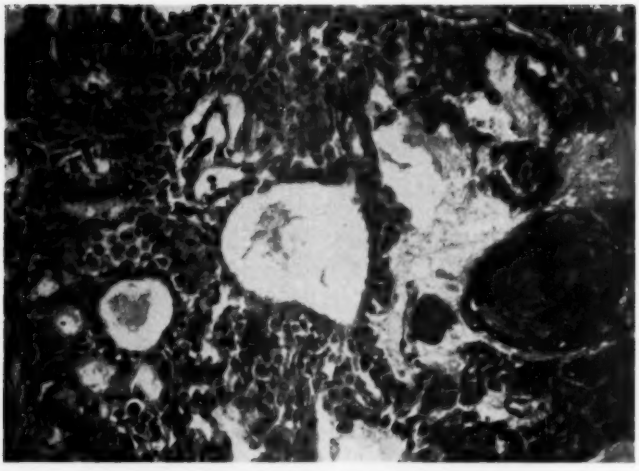

Fig. 4.—Adenoid cystic epithelioma x 220. Note the epithelial cells, the gland-like spaces and the epithelial pearl, and the mucoid material.

arise from another mother cell, which is the basal cell of the epidermis or the skin appendages.

*Diagnosis.* Multiple growths especially when seen early look like cystic tumors of sweat glands called syringomata or hydradenomata and multiple sebaceous adenomata. Syringomata, however, are smaller and appear more on the chest. The face is commonly affected in the other two types. They can, however, be easily differentiated histologically. This is important because tumors of the sweat glands are less apt to be familial and are radiosensitive while cystic epitheliomata are radioresistant.[14] The tumors have also to be differentiated clinically from nonpigmented moles, multiple neurofibromata and lipomata especially when small. The solitary type is still more difficult to differentiate clinically from other tumors. In fact, Case 1 was diagnosed clinically as chondroma or fibroma.

*Nature.* Before closing we should comment on the nature of the tumor, whether it should be considered benign or malignant. Many authors including Willis (1948) believe that this disease is benign. They stressed their belief by adding the prefix benign to the name of the tumor. Patients with such multiple growths have been followed for years (up to 20) with no malignant change. Malignancy has been

described by other authors following irritation or improper treatment. Streitman (1946) reported a case with metastases in a supraclavicular lymph node which appeared 15 years after the primary growth. Willis designated this latter as a secondary growth because it appeared after such a long time. Also there was no lymphoid tissue in the section around the supraclavicular growth. He rightly concluded that this was probably another tumor unusually situated at the site of a lymph node. Olive Gates[13] found that among 116 cases reported in the literature in detail there were 5 cases (4.3 per cent) of secondarily arising carcinoma usually of the basal cell type. Louis Stavard[7] examined an affected family for three generations. He found in 1938 nine persons developing the disease and one of them developed an epithelioma on the forehead which the patient said had developed from one of the little tumors, which has been growing for 12 months. The same patient also developed an abscess in a keratin cyst with foreign body giant cells but no tumor tissue apart from the cyst.

From our study we believe that the multiple lesion may be considered an abnormality in development rather than true neoplasia and is mostly benign. The solitary lesion is, however, neoplastic and may be locally malignant. Our case of solitary tumor of the nose, although it was clinicaly benign and easily enucleated, was active histologically and infiltrated the capsules.

SUMMARY

The nature, origin and pathology of adenoid cystic epithelioma of both multiple and solitary type are discussed together with report of an example of each.

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LXXVIII

# SARCOMA OF THE HEAD AND NECK IN CHILDHOOD

H. Markowicz, M.D.

AND

E. Shanon, M.D.

Tel Aviv, Israel

Malignant neoplasms are an important cause of death in childhood. This became more evident during the past decade as the role played by infectious diseases as causes of death decreased markedly.[1] In the Children's Memorial Hospital (New York) up to the year 1947 there were among 730 autopsies in children aged 12 years or less 103 (14.1%) malignant tumors and among 97 autopsies in children from 13 to 16 years inclusive 30 (30.9%) malignant tumors.

Sarcoma appears to be much more common than carcinoma in childhood. Out of 219 malignant tumors in children aged 12 years or less, reported by Steiner,[2] there was only one carcinoma (in a liver showing juvenile cirrhosis). Lymphatic and hemopoietic malignant tumors, neuroblastomas, intracranial and Wilm's tumors were the most common forms of fatal malignant neoplasms in childhood, the incidence of sarcoma ranging from 3.9% (University of Chicago series) up to 6% (Children's Memorial Hospital).

New and Hertz[3] analyzed 235 cases of malignant diseases of the face, mouth, pharynx and larynx occurring in the first three decades of life (Mayo Clinic, 1907-1932). The incidence of sarcoma in the whole group was 12%.

The present paper deals with seventeen cases of sarcoma occurring in the head and neck in children aged 3½ to 13 years. These cases were observed during a six years period (1952-1958) in the Head

From the Head and Neck Service, Department of Otolaryngology, Beilinson Hospital, General Federation of Jewish Labour in Israel, Sick Fund (Kupath-Holim), Petah-Tikva, Israel.

and Neck Tumor Clinic. The material is presented according to anatomical regions.

## REPORT OF CASES

### *I. Sarcoma of the Nasopharynx*

CASE 1. M.J., a four year old boy born in Israel, was referred to our department with the diagnosis of a retropharyngeal abscess. His family and past history were essentially negative. Two months before hospitalization he started to breathe with his mouth open. The respiratory disturbance became worse in the course of a few weeks, enlarged nodes appeared in the neck on the left side, and the temperature rose to 39° C. A retropharyngeal abscess was suspected and antibiotics were administered. As the child did not improve he was admitted for incision and drainage of the abscess.

A marked depression of the soft palate and peritonsillar swelling were seen on the left side. A large soft tissue mass could be palpated in the nasopharynx and enlarged, hard, polycyclic nodes in the neck on the left side. Smaller nodes were present on the right side as well. A tumor was suspected and a biopsy of a cervical node revealed a lymphosarcoma. Deep x-ray irradiation was given (3714 r, 200 KV, 2 fields). The immediate reaction was favorable and the tumor mass receded rapidly. However, the growth recurred after a short time and the child died two months after termination of radiation therapy. A postmortem examination was not performed.

CASE 2. E.L., a 12 year old girl born in Iraq, resident in Israel for 2½ years, was admitted because of sore throat and fever. Her family history was negative. For several years she had suffered from recurrent spells of cough, especially during the summer months. Three months prior to admission enlarged lymph nodes appeared in the neck on both sides. Fluoroscopy revealed several small calcified foci in both lungs. E.S.R. was 97 mm in the first hour (Westergren). Tuberculous adenitis was diagnosed and she was given ambulatory radiotherapy and nicotibine for several months.

On admission acute tonsillitis was diagnosed and a large mass in the nasopharynx was found. Biopsy of the cervical node showed a reticulum cell sarcoma. Intensive deep x-ray therapy (7008 r, 200 KV) was given over cervical, temporal, frontal and occipital fields. The tumor disappeared gradually and the girl remained well up to date, four years after termination of the treatment.

*Comment.* Malignancy of the nasopharynx may occur at any age, but the maximal incidence is in patients over 40. However, it is noteworthy that out of the 25 cases observed by us during the past six years, nine were less than 40 and six less than 30 years of age. Besides the two described cases of sarcoma we also encountered one case of squamous cell carcinoma of the nasopharynx in a boy aged 14, which was irradiated; the patient is alive and well five years later. Ward and Hendrick[4] reported the incidence of sarcoma in the nasopharynx as 45% and that of carcinoma 55%. Sarcoma is more prevalent in the first decade of life and it invades cervical nodes earlier than carcinoma.

The importance of routine examination of the nasopharynx is clearly demonstrated in our second case in which antituberculous therapy was given for several months. Before incising a peritonsillar abscess in children or in older patients, the possibility of a malignant tumor masquerading as an abscess should be kept in mind.

The prognosis of malignant neoplasms of the nasopharynx is poor especially in the younger age group. A survival period of five years has been reported in 25% of cases, or less. Recurrent cases are usually resistant to irradiation but recently successful attempts have been made to treat such cases with a combination of electrodessication, radioactive cobalt and x-ray radiation.[5]

### *II. Sarcoma of the Nose and Paranasal Sinuses*

CASE 3. L.T., a five year old girl born in Tripoli, resident in Israel for one year. Three months before admission a fleshy mass was observed in the left nasal cavity. This was repeatedly incised. On admission the left nasal cavity was found to be occluded by a tumor mass; an external swelling was also visible in the upper part of the nasal bone on the left. X-ray examination showed clouding of the left maxillary and ethmoid sinuses but no signs of bone destruction. Biopsy from the nose revealed a sarcoma. Deep x-ray radiation was given: 4000 r over three fields, followed by a second series of 2000 r over two fields, three months later. The girl is well and free of disease nine years after termination of treatment.

CASE 4. S.L., a four year old girl born in Rumania, resident in Israel for one year, was admitted with the diagnosis of a brain tumor. Her family and past history were not relevant. One and a half months before hospitalization she complained of right ear- and eye-

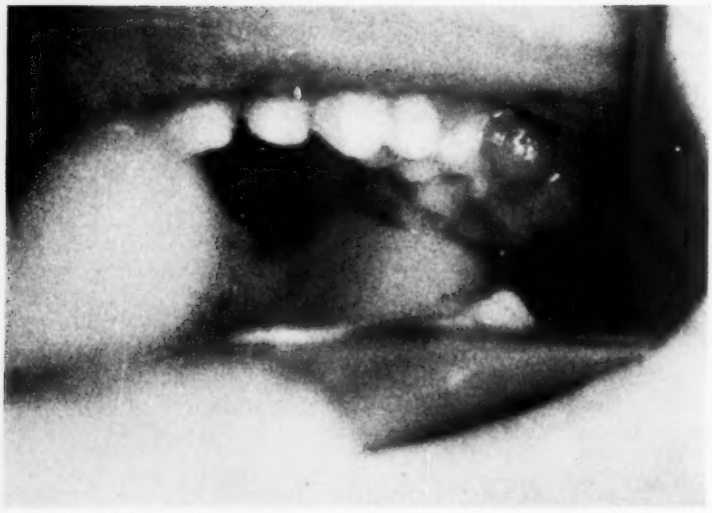

Fig. 1.—Lymphosarcoma in the region of the left upper bicuspid.

ache. An examination in another hospital showed paresis of the right oculomotor and abducent nerves.

On admission to our department the temperature was 39.8° C. Roentgenological examination revealed an opacity of both maxillary sinuses; the foramen ovale was indistinct on the right side. Osteomyelitis of the right maxilla was suspected and intensive antibiotic treatment was started (penicillin, aureomycin). Marked improvement followed, the temperature fell, free motility of the eyeball returned, the maxillary swelling and the nasal discharge disappeared and the ESR became normal. However, one month later blindness supervened in the right eye, the protrusion increased markedly and optic atrophy was found.

The ESR was 50/85, routine blood chemistry, serology and C.S.F. were normal.

A paresis of the III, VI and V cranial nerves was present on the right side. Tongue and uvula deviated to the left. The right eyeball protruded. The eyegrounds were normal. Edematous swelling was seen over the right maxillary and zygomatic regions. There was

purulent discharge from the right nasal cavity. The patient began to complain of pain over the right maxillary region.

Surgical exploration was made six months after the onset of the illness. Through an incision along the right lateral nasal wall the anterior ethmoidal cells and the frontal sinus were opened and were found to be unaltered. On opening the right maxillary sinus, a smooth, dark-red tumor, the size of a plum was found, originating from the posteromedial wall. The tumor was partly removed and erosion of the posterior sinus wall could be seen. Apparently the mass grew from the posterior ethmoidal cells into the maxillary sinus and into the pterygomaxillary fossa. The pathologist diagnosed a small cell sarcoma. The tumor was irradiated (4400 r) but the child's condition deteriorated rapidly and she died three months after operation. Post-mortem examination was not performed.

Case 5. C.S., a six year old boy born in Israel, was admitted with a swelling of the gingiva. His family and past histories were essentially negative. A fortnight before admission the boy complained of toothache, his temperature was found to be elevated and a swelling was observed in the region of the second left upper bicuspid. The tooth was extracted but the swelling increased in size and the child complained of pain over his left maxilla. On admission a reddish grey soft mass was seen in the region of the extracted tooth (Fig. 1). The hard palate was depressed on the left side. X-ray examination showed opacity of the left maxillary sinus as well as destruction of its roof and lateral wall. Biopsy revealed a lymphosarcoma. Radiotherapy was given (3520 r, 250 KV) and the tumor disappeared. However, the general condition of the child deteriorated rapidly and he died a week after termination of treatment. Autopsy revealed generalized lymphosarcomatosis involving both kidneys, para-aortic and mesenteric lymph nodes, stomach, liver, pancreas, vertebrae, heart and dura mater.

*Comment.* Cade[6] differentiates three groups of sarcomata in the nose and nasal sinuses: the low-grade malignant tumors (giant cell tumor, angiosarcoma, myxosarcoma), the medium-grade malignant tumors (fibrosarcoma and chondrosarcoma), and the high-grade malignant tumors (round-cell sarcoma, comprising reticulosarcoma, lymphosarcoma and Ewing's tumor).

Of 29 cases of sarcoma reported by Harmer one half were alive for more than five years.

Fig. 2.—Neurofibrosarcoma of the orbital tissues growing through the nasal cavity.

### *III. Sarcoma of the Orbital Tissues*

CASE 6. L.D., an eight year old boy born in Israel. In December 1953 the mother noticed a mass in the lateral part of the left eye. Four months later the child was admitted to an eye department in another hospital. The tumor was excised together with the eyeball. It was apparent that the mass grew from the orbital tissues. The pathologist diagnosed a neurofibrosarcoma. The tumor recurred and deep x-ray irradiation was given as well as another operation performed. The tumor continued to grow and invaded the nasal cavity (Fig. 2). Severe hemorrhages followed. The child died three years after the onset of the disease.

### *IV. Sarcoma of the Tonsil*

CASE 7. T.D., a four year old Israeli boy, had been suffering from recurrent bouts of acute tonsillitis. Three months prior to admission an ear, nose and throat specialist observed an enlargement of the left tonsil and recommended tonsillectomy. The operation was postponed because of a cold. The left tonsil increased markedly in size and the child was referred to our department. On examination the left tonsil was found to be the size of a plum, bluish-red, firm and bled easily on touch. Biopsy showed a lymphosarcoma. The child was treated by deep x-ray irradiation (4800 r, 200 KV). The tumor regressed rapidly and the child remained healthy and well up to date five years after therapy.

*Comment.* Lymphosarcoma constitutes 18% of malignant tumors of the tonsil.[4] In this series it represented one out of eight miscellaneous malignant tonsillar growths that were observed during the past five years, most of the cases in an elderly age group. Lymphosarcoma though highly radiosensitive has a high rate of recurrence and tends to produce early local and distant metastases. An asymmetrical enlargement of tonsils should be viewed with suspicion and a biopsy must be preceded by irradiation to prevent a possible spread of the tumor. The favorable results of radiotherapy in our case of lymphosarcoma of the tonsil shows that statistical data should not have a determining influence on our attitude and prognosis in the individual cancer case.

### *V. Sarcoma of the Pyriform Sinus*

CASE 8. B.J., a three and one-fourth year old girl born in Israel, was examined by an ear, nose and throat specialist because of a cold, five days prior to hospitalization. A tumor mass was seen in the oropharynx.

On admission the child's general condition was poor. She looked pale and breathed with her mouth open. A round mass, the size of a strawberry, deep red and smooth, was observed behind the base of the tongue. Small, soft lymph nodes were palpable in the neck, in both submaxillary triangles. Examination of the chest revealed no pathological findings. Liver was palpable one and a half finger breadths below the costal arch; no splenic enlargement was discernible. The child recovered from the cold following symptomatic treatment. She was discharged from the hospital to be readmit-

ted for further study three weeks later. Her general condition grew worse. She appeared restless and suffered from recurrent spells of dry cough at night.

Routine laboratory studies of blood and urine revealed values within the normal range. Fluoroscopy of the chest showed no pathological findings. A round mass at the base of the tongue could be seen on a lateral x-ray film. Uptake of radioactive iodine was normal over the thyroid region. Some uptake was also disclosed over the tumor, but it could not be decided whether this was due to radiation from the thyroid gland. A tentative diagnosis of a true accessory thyroid was made. Surgery was resorted to, the intention being to excise the mass under general anesthesia with endotracheal intubation. However, repeated attempts at intubation failed as the tumor partly occluded the laryngeal entrance. The child received pentothal-curare as premedication to intubation and despite the administration of oxygen her respiration appeared markedly depressed. Thus, the decision was made to remove the tumor as rapidly as possible using a laryngoscope and to intubate thereafter. The mass was grasped, encircled by a snare and removed. Brisk bleeding followed; suction was carried out and clamps were applied to the bleeding area. The child turned blue and breathing stopped. Tracheotomy was performed immediately and oxygen was given. However, cardiac arrest supervened. Fifteen minutes elapsed until cardiac massage was started. The heart resumed its normal beat. Body temperature was brought down to 32° C, using ether and ice-packs, and largactil administered intravenously. Respiratory movements reappeared, but noradrenaline infusion had to be given constantly to keep up the blood pressure. Twelve hours after restitution of the cardiac beat the kidneys began to function. Twelve hours later spinal reflexes appeared, but unconsciousness persisted. Seventy-two hours after the operation the child died.

On post-mortem examination the brain was bluish-red, of a soft consistency. Numerous small hemorrhages were seen in the white substance. A white rounded tumor the size of a small cherry was found in the right pyriform sinus. The tumor infiltrated the hypopharyngeal wall (Fig. 3). The thyroid gland was unaltered. The left lung was atelectatic. The heart was normal. The liver and the spleen showed no gross pathological changes. The retroperitonal tissues were markedly edematous. The kidneys appeared normal.

Microscopic examination revealed a highly undifferentiated sarcomatous tumor originating from the wall of the pyriform sinus. In

Fig. 3.—Part of the tumor (undifferentiated sarcoma) in the pyriform sinus.

the brain degenerative neuronal changes were seen, especially in the cerebellum and in the basal ganglia. The nuclei appeared shrunken and rich in chromatin Nissl's granules were usually missing. Thrombotic changes were observed in the lungs. The heart showed cellular perivascular infiltration in a few places. Inflammatory changes were present in the adrenal cortex. The kidneys appeared normal except for a few recent thrombi.

*Comment.* Sarcoma of the hypopharynx and esophagus is known to occur in two forms: polypoid and ulcerative. The polypoid sarcoma is usually well circumscribed and has little tendency to metastasize. At times the growth assumes a pedunculated appearance. The ulcerative form grows more rapidly and metastasizes widely.[8] Lotheisen[9] analyzed 38 cases of esophageal sarcoma and there were six

Fig. 4.—Undifferentiated sarcoma of the middle ear. The tumor bulges out of the external auditory meatus.

females among them. Albrecht[10] described a case of sarcoma in the lower part of the pyriform sinus and Kohl[11] observed a spindle cell sarcoma in the hypopharynx. Stephen[12] reported a case of lymphosarcoma in the lower part of the esophagus in a four year old boy. We have observed a case of a pedunculated sarcoma in an adult male. The tumor originated from the lateral wall of the hypopharynx. Following excision by a snare and deep x-ray irradiation the patient is alive and well five years after termination of treatment. It may be remarked that the pedunculated form of sarcoma is well known to occur in the larynx. The point of attachment is usually not extensive, the tumor is relatively avascular and has little tendency to infiltrate the laryngeal structure. Hence, at times it can be removed readily without sacrifice of the larynx (snare, biopsy forceps, thyrotomy).[13,14]

Our case showed several peculiarities; it masqueraded closely as a lingual thyroid. Though pedunculated, the tumor infiltrated deeply into the hypopharyngeal wall assuming the form of an hour-glass, and its vascularity was marked. No doubt an elective tracheotomy was indicated in this case. Partial excision of the growth followed by radiotherapy would have probably prolonged the child's life.

### *VI. Sarcoma of the Ear*

CASE 9. F.E., a five and one-half year old female child born in Israel. Her family and past history were irrelevant. One month prior to admission the child complained of a left earache. The pain subsided following penicillin therapy but it recurred after a week's time. A serofibrinous aural discharge appeared and an ear, nose and throat specialist diagnosed a furuncle of the external meatus. A fortnight before the admission brisk bleeding appeared from the diseased ear. The meatus filled up rapidly with polypoid mass and the periauricular tissues swelled up markedly. Pain, trismus and difficulty in moving the head followed.

On examination a dark red tissue mass was seen bulging out from the left external auditory meatus (Fig. 4). Marked swelling and possibly fluctuation of the tissues behind and below the auricle were observed. The area was markedly tender to touch. The child kept its head to the left side as in torticollis. Physical examination of other regions, routine laboratory studies of blood and urine, and an x-ray examination of the chest revealed no pathological findings. Radiologic studies of the mastoids showed normal pneumatization on both sides with a slight opacity on the left. Mayer's projection revealed a localized area of destruction in the antero-inferior external meatal wall. X-ray of the base of the skull showed destruction of the annulus tympanicus and of the anterior meatal wall. Auditory ossicles could be recognized on both sides.

Biopsy showed an undifferentiated sarcoma. Deep roentgen therapy was started and 4000 r (250 K.V., 3 fields) were given over a period of three weeks. The bulky tumor mass diminished in size and the peri-auricular swelling disappeared. However, a control biopsy showed the sarcomatous growth persisting in the external meatus.

The child's general condition deteriorated rapidly. She complained of severe abdominal cramps. Osteolytic metastases were dem-

Fig. 5.—Sarcoma of the middle ear destroying the cranial base.

onstrated roentgenologically in pubis, ischium and femur on both sides. The child died three months after the treatment was started. A post-mortem examination was not done because permission was not granted.

CASE 10. G.N., a four year old Israeli boy. His family and past history were non-contributory. The onset of the illness, five months previously, was marked by the appearance of aural polyps and a purulent, blood stained aural discharge with little pain and low grade fever. The polyps were excised twice but recurred soon after. Because of this the child was referred to our out-patient department.

On examination a polypoid mass was found filling the left external auditory meatus. On studying the x-ray films it was surprising to find an undisturbed pneumatization in the diseased ear; this seemed inconsistent with the diagnosis of a chronic inflammation.

A biopsy revealed sarcoma with numerous mitoses and atypical pleomorphic cells. An x-ray examination of the base of the skull showed destruction of the anterior half of the left petrous pyramid. A peripheral left facial palsy developed.

Deep x-ray treatment was given—4800 r (250 K.V., 2 fields) in three and a half weeks. The facial palsy regressed promptly and the tumor mass decreased in size. However, three months later gross recurrence of the tumor was observed and paralysis of the III, IV, VI and VII cranial nerves followed. The child died about 17 months after the onset of his illness.

On post-mortem examination a considerable amount of soft gelatinous grey tissue was seen filling the middle ear and the auditory meatus, destroying the cranial base and invading the left orbit and the ethmoid sinuses (Fig. 5.). No significant pathological changes were observed elsewhere.

*Comment.* Sarcoma of the external ear is rare. Among 143 malignant tumors of the external ear reported by Boland and Paterson[15] (Christie Hospital, Manchester, 1932-1955) there was not one case of sarcoma. Loch[4] described sarcoma of the ear as a round, well defined tumor with a smooth surface and a rare tendency to metastasize, occurring usually in middle age. Fibrosarcoma, osteosarcoma and angiosarcoma have been reported.[16]

Sarcoma of the middle ear may be found in all age groups but is more frequent in the first decade of life.[4] It may perforate to the outside or to the endocranial cavity. Osteosarcoma and melanosarcoma have been described. Fibrosarcoma was described in a child seven years old and rhabdomyosarcoma in a three year old one.[16]

Our two cases of sarcoma involving the ear were encountered among four malignant tumors of the ear (auricle excluded) observed during the past six years (the other cases being a hemangio-endothelsarcoma of the external meatus in a woman aged 22 years, and a squamous cell carcinoma of the middle ear in a male patient 44 years old).

The chief diagnostic points suggestive of a malignant tumor were, in our cases, bleeding and rapid recurrence of polyps following excision. The radiologic demonstration of good pneumatization of the mastoid process in a case of clinical chronic otitis media should be viewed with suspicion. It is evident that polypoid tissue issuing from the ear should be examined histologically more often, particularly if it tends to recur.

### *VII. Sarcoma of the Skull and Jaws*

CASE 11. G.H., a four year old girl, was admitted to our hospital because of a swelling in the frontal region of two months' duration.

Fig. 6.—Ewing's sarcoma of the jaws.

There was no pain but body temperature was elevated. Bilateral papilledema was found. Neurological examination revealed no pathological findings. X-ray examination showed areas of destruction in the frontal bone with irregular borders. Translucent areas were also observed in the upper part of the right humerus and tibia. Laboratory studies of blood, urine and CSF showed normal values. A tissue specimen was obtained from the frontal region and the pathologist diagnosed Ewing's sarcoma. Deep x-ray therapy was given (2000 r, 200 K.V., 2 fields over the skull). The child failed to come for further therapy and died three months later. No post-mortem examination was done.

CASE 12. S.D., a 12 year old boy born in Tripoli and resident in Israel for five years. His family and past history was non-contribu-

tory. The present illness started two months prior to admission with the appearance of a small prominence in the vicinity of the first left lower tooth. This increased gradually in size but caused no pain. On examination a nodular elastic mass was found covered by gingival mucosa and non-tender to pressure. X-ray examination revealed a cystic structure with destruction of the compact bone and signs of new bone formation. On surgical exploration a pale, rosy, fleshy tumor mass was found covered by thin cortical bone. It was firm and well delimited and could be easily enucleated. The adjoining tooth was extracted and the adjacent bone curetted out.

Biopsy showed an osteogenic sarcoma.

4340 r were given (250 K.V.) over two mandibular fields. Two years later there were no signs of local recurrence.

Case 13. K.D., an 11 year old boy, born in Israel. His family and past history were essentially negative.

Three months prior to admission to our department, the boy fell on his back and complained of pain in both lower extremities. In another hospital, a complete general and neurological examination disclosed no abnormal findings. Three weeks before admission, a swelling appeared over both canine fossae. The child complained of loose teeth. In another hospital sinusitis was suspected and, later, the possibility of lead poisoning was considered. However, this was not confirmed by laboratory examination.

The child was transferred to our department for further study.

He was found to be a well-developed, rather obese boy in a good general condition. A marked bulge was observed over both canine fossae (Fig. 6). Marked swelling and erosions were seen around the teeth (Fig. 7). The jaws and gingivae were tender to touch. The nose, nasopharynx and ears appeared unaltered. A few, small, discrete nodes were palpated in the neck on both sides. Examination of the chest, abdomen and extremities disclosed no abnormal findings except for numerous light brown spots on the skin of the face, chest and extremities.

Temperature was normal. E.S.R.—18/41, blood count, blood chemistry and urine examination were normal.

X-ray study showed destruction of the lateral wall of the maxillary sinus on both sides as well as osteporotic foci in the mandible. X-ray of the chest and of the long bones showed no abnormality.

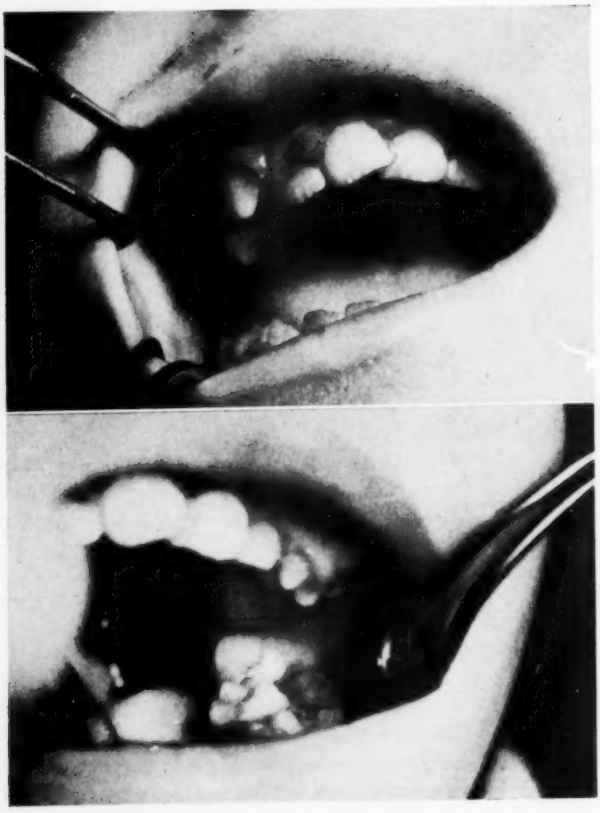

Fig 7.—Ewing's sarcoma: gingival lesions.

The left maxillary sinus was explored under local anesthesia. After incising the mucosa, a mass of vascular soft tissue was found filling the sinus. The anterior wall of the sinus was missing. Biopsy revealed Ewing's sarcoma.

Deep x-ray therapy was started: three fields (right, left, anterior). The intended dose (air) is 8-10-200 r over each field (200 K.V.).

The immediate response to irradiation was remarkable, the tumor melted away. However, an x-ray examination of the pelvis showed osteolytic foci in the ischium and pubis on the left. A soft mass was seen in the right orbit, just above the eyeball. The child still receives deep x-ray therapy.

*Comment.* Osteogenic sarcoma constitutes about one half of bone tumors in childhood.[7] In Geschickter and Copeland's series[17] of osteogenic sarcoma (sclerosing and osteolytic) the maximal age incidence was 10 to 25 years; 35 out of 187 cases of the sclerosing form were 5 to 15 years old. The upper and lower jaws were involved in 26 out of more than 700 cases of primary bone sarcomas and the cranial bones were involved in 12. The radiologic picture of the osteolytic form was often erroneously interpreted as a benign cyst and benign giant cell tumor. Although on the whole the prognosis of these tumors was poor, 16% of the osteolytic group and 25% of the sclerosing group surviving five years, cases were known which were characterized by a slow rate of growth. Ewing[7] described patients who survived 21 and 25 years. Trauma proved to be a factor accelerating a slow growing tumor. Ward and Hendrick[4] recommended radical surgery as the treatment of choice, sarcoma of bone being as a rule radioresistant. Their patients lived six months to two years after diagnosis was made. Geschickter and Copeland[17] also advised prompt resection and permanent cures were attained by this method, e.g., a case was cited of a girl 13 years old with osteogenic sarcoma of the mandible, who remained well 25 years following surgery.

The decision to perform an extensive, mutilating jaw resection is a very difficult one to make, particularly so in the case of a child. It is thus natural that all of us follow expectantly the recent reports of Sir Stanford Cade on the value of supervoltage irradiation as an effective means of controlling bone sarcomas.[18]

### *VIII. Hodgkin's Sarcoma*

CASE 14. S.I., an eight year old boy born in Israel, was treated for the past two years with small, repeated doses of x-rays, because of enlarged cervical nodes on the right which were considered as tuberculous in origin. The nodes increased gradually in size and on admission to our department the cervical mass of nodes measured 7x10 cm. The mass was excised and the pathological diagnosis was Hodgkin's sarcoma. Postoperative irradiation, 900 r, 200 K.V., was given. Two years later hepatosplenomegaly and a mediastinal mass were observed. 2400 r (200 K.V.) were given over the spleen and 1744 r (250 K.V.) over the mediastinum. The immediate response was favorable. The boy is alive six years later, but during this time enlarged nodes appeared and were irradiated in the neck, axillae and inguinal regions.

CASE 15. B.M., a boy aged seven and a half years. One year prior to the present admission a node appeared in the right cervical region. The node grew in size and several other nodes were observed as well. Biopsy revealed a Hodgkin's sarcoma. Neck dissection was performed on the right side preserving the sternomastoid muscle. This was followed by irradiation (900 r, 200 K.V.). Three years later the cervical nodes recurred on the right side and were irradiated again (1500 r, 200 K.V.). No other foci of the disease could be demonstrated in other parts of the body, and he is well four and a half years after the operation.

CASE 16. S.S., a boy aged 12 years. For four months before the present admission enlarged cervical nodes were observed on the right side. The tentative diagnosis was cervical lymphadenitis of unknown etiology and the treatment was a symptomatic one. On admission a chain of lymph nodes the size of a pea to a cherry was found in the anterior cervical triangle on both sides. Biopsy showed a Hodgkin's sarcoma. 1600 r (200 K.V.) were given to each side of the neck. The nodes disappeared and the child's condition was satisfactory for the following six months. No pathological foci could be discerned elsewhere in the body, for two years, but his general condition deteriorated markedly.

CASE 17. P.S., a 13 year old girl born in Israel, was admitted to our department because of a low grade fever of three months' duration. Enlarged nodes were palpated in the supraclavicular region on both sides and on an x-ray examination a widening of the mediastenal shadow was found on the right side. Biopsy of a cervical node

showed a Hodgkin's sarcoma. Following a therapy with nitrogen mustard a node persisted in the left supraclavicular region. This was irradiated (1500 r, 200 K.V.). A year and half after the treatment a small node was found anterior to the right sternomastoid muscle. It will be excised for pathological study.

*Comment.* Our limited experience does not permit an evaluation of neck dissection for Hodgkin's disease apparently localized to the neck on one side.

## SUMMARY

Seventeen cases of sarcoma of the head and neck in children, 11 boys and 6 girls, are described. The average age was 7.4 years and the average duration of symptoms before the diagnosis was 4.3 months. Only five cases out of the whole group were referred to us with a tentative diagnosis of a malignant neoplasm. Failure to make the correct diagnosis was due to incomplete examination, for example, omission of posterior rhinoscopy, or false clinical assumptions not backed by biopsy, (for example, omission in the cases of Hodgkin's sarcoma and in sarcoma of the ear). Clinically the tumor closely simulated such conditions as retropharyngeal abscess, tuberculous lymphadenitis, lingual thyroid, furuncle of the external auditory meatus, chronic otitis media with polyps, radicular cyst and osteomyelitis of the jaws. In addition to the five cases referred to us as tumors, in eight others a pre-operative diagnosis of a malignant neoplasm was seriously considered. However, in four cases the real nature of the disease was not suspected until a biopsy was made (sarcoma of the pryiform sinus, sarcoma of the middle ear, osteogenic sarcoma and Ewing's sarcoma of the jaws). In eleven cases irradiation only was resorted to, in five surgery and irradiation and in one surgery only. Seven children died within 12 months after treatment (six died within three months). One died two and a half years after treatment. Three are alive with recurrence of the disease (Hodgkin's sarcoma) one and a half, two and six years after termination of treatment. A child with Ewing's sarcoma of the jaws is still irradiated. One case of Hodgkin's sarcoma and one of osteogenic sarcoma are alive and well two to four years after treatment. One child (reticulum cell sarcoma of the nasopharynx) is alive and well four years and another (lymphosarcoma of the tonsil) four years after treatment. A child treated for sarcoma of the nasal cavity is alive and well nine years later.

The relatively small number of the cases and the short follow-up period do not allow to draw generalizations and conclusions. This

series indicates that an early diagnosis may not be easy and tumor alertness as well as frequent resort to biopsy appear to be the most efficient factors in establishing the true nature of the disease. The value of mutilating operations on children suffering from sarcoma of the head and neck is questioned. Future results of supervoltage irradiation are awaited with hopeful expectation.

13 MENDELE ST.

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LXXIX

# MIDDLE EAR MUSCLE ACTIVITY AT MODERATE SOUND LEVELS

F. Blair Simmons, M.D.

Stanford, Calif.

The role of the middle ear muscles in everyday audition has apparently received little emphasis in the past. A review of the literature leaves one with the impression that the tensor tympani and stapedius muscles are at best interesting masses in the middle ear, inert unless very loud sounds are presented; that with the introduction of loud sounds (80-90 db) these muscles suddenly spring to life, contracting and reducing the sound amplitude reaching the cochlea.

In spite of the fact that thresholds in decerebrate, strychninized animals have occasionally been reported as low as 40 db under optimal circumstances,[1,2] the prevailing opinion seems to be that of Wever and Lawrence[3] who said: "It is evident that for the greater part of the auditory range the muscle reflexes occur only when the stimulating tones rise to a high level of intensity."

This attitude may have developed because of studies done primarily on anesthetized or decerebrate animals (cat, guinea pig, rabbit). In such studies it has been well established that the primary result of middle ear muscle contraction is reduction of cochlear microphonic amplitude. Possibly because these contractions are stronger at higher sound intensities, it has been assumed that the only function of these muscles is protection against acoustic trauma. While this author has no quarrel with the trauma concept, it is possible that other functions do exist. Indeed, it will be the primary purpose of this paper to demonstrate the importance of the middle ear muscles in hearing at moderate sound levels.

## METHODS

This investigator became impressed with the effects of middle ear muscle activity at moderate sound levels while participating in several

From the Department of Neurophysiology, Walter Reed Army Institute of Research, Washington, D. C.

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of the experiments of Galambos and Rupert.[4] These experiments involved the recording of cochlear potentials of awake cats through electrodes permanently implanted on their round windows. These cochlear potentials, and especially the cochlear microphonic, showed marked changes resulting from both sound-induced and spontaneous contractions of the middle ear muscles. In many ways, the experiments described below are an extension of that work.

The experimental procedures were, for the most part, based on the following premises:

1. The amplitude of the cochlear microphonic recorded at the round window is, among other things, directly proportional to the sound intensity transmitted to the cochlea across the middle ear.

2. In animals this ossicular transmission is altered by contractions of the middle ear muscles.

3. There are no other significant *dynamic* factors which influence this transmission, or the amplitude of the cochlear microphonic.

4. The effects of the middle ear muscles can be eliminated permanently, by cutting, or temporarily, with anesthesia.

5. The difference observed between the amplitude of cochlear microphonic in a normal awake cat (tensor and stapedius active) and that in the same animal when anesthetized (tensor and stapedius inactive) is equal to the influence of the two muscles.

Verifications of Statements 1, 2, and 4 are available in the previously cited literature. The following procedures were carried out to verify Statement 3 and to study the effects of the middle ear muscles in normal, awake cats.

Two groups of normal adult cats were operated upon using sterile technique. With the aid of a dissecting microscope, a stainless steel electrode was implanted on the round window of each cat. For stability, the distal portion of this electrode was firmly anchored to the posterior wall of the bulla before being externalized at the vertex of the skull. In one group of these cats the middle ear muscles were left intact. In the other, both the tensor tympani and stapedius muscle tendons were cut at their insertions without damaging the ossicular chain. Most animals were implanted bilaterally. Usually the type of muscle preparation was randomized so that an animal had a normally muscled ear on one side and a demuscled ear on the other.

After surgery, the cats were given antibiotics and random-length recovery periods. No animal was tested until at least ten days after surgery; most were not evaluated for two or three weeks. At the end of that time, cats having cochlear potentials within the normal range were placed in the study, while the sub-standard animals were discarded.

The stimulus tones were generated in a General Radio audio oscillator and passed through a 10 db step attenuator into a hearing aid receiver (Audivox 9-C). This receiver was imbedded in a cat ear-mold which fit snugly in the cat's external auditory canal. This closed-field sound source obviated the effects of cat movement or any major disturbance in the sound field. Unfortunately, the use of this type of transducer limited somewhat the intensity and frequency ranges. Frequencies below 0.2 and above about 9 Kc were not accurately reproduced. Those between these limits were stable and of good quality at the intensities used. At the highest level generated (about 90 db) the second harmonic was down more than 20 db. Also the frequency-intensity output was not uniform, as can be surmised from Figure 1. Consequently, the results presented are limited to the frequencies between 0.5 and 7 Kc where the isointensity curve, as related to human threshold, varied $\pm$ 5 db from its value at 1 Kc. A 2 cc coupler and Allison artificial ear were used to calibrate the sound source. Periodic checks with the artificial ear and by subjective methods were made throughout the experimental period. It is believed that over-all calibration error did not exceed 5 db.

The sound used to produce cochlear microphonic and to stimulate middle ear muscle contractions were of two types. One was a continuous pure tone; the other, a swept pure tone similar to that of a Bekesy audiometer. Microphonic responses to these stimuli and dynamic response changes were recorded, after suitable amplification, on a Sound Apparatus Company Polinear Frequency Recorder calibrated to write in voltage of cochlear microphonic. When swept tones were used, mechanical linkage of the recorder and audio oscillator through a common drive motor made possible a continuous plotting of cochlear microphonic amplitude with changing frequency. In most instances where a swept tone was the stimulus, measurements were made from 0.2 through 10 Kc. The cochlear microphonic response to continuous single-frequency tones was recorded at 0.5, 0.7, 1, 1.5, 2, 3, 5 and 7 Kc. Serial recordings were made in 10 db steps of ascending or descending intensity.

The over-all sensitivity of the recording system was reliably better than 10 uv (RMS). The signal amplification was, however,

subject to about 3 db of drift on occasion. Drift and sensitivity were checked periodically with a General Radio Microvolter, and occasionally with a cathode ray oscilloscope.

To measure middle ear muscle activity as reflected in the cochlear microphonic, the fully awake test animal was placed in a restraining box with his head free to move. After careful insertion of the ear mold and connection of the round window electrodes, either or both types of stimulating tones were presented in a systematic manner. Immediately upon completion of the awake portion of the testing, the animal was anesthetized with nembutal, either intraperitoneally or intravenously. After deep anesthesia was obtained, the same set of acoustic stimuli were presented and cochlear microphonics recorded in a manner identical to the awake procedure.

As a control measure with intravenous anesthesia, the animals were prepared beforehand with an indwelling polyethylene catheter which led outside the restraining box. Thus, the cat could be anesthetized without disturbing any portion of the experimental environment.

## RESULTS

The cochlear microphonic responses of a demuscled ear to the testing situation are illustrated in Figure 1. These recordings of cochlear microphonic amplitude were first obtained from the awake cat (solid lines) by exposure to the swept-frequency tones at three intensities. These same three levels were then repeated approximately 15 minutes later when the cat was deeply anesthetized (dotted lines). The difference between the microphonic responses in the two sets of measurements is negligible. The slight variation that does occur is within the drift limits of the amplifier, except at one point between 1.7 and 2.7 Kc which will be discussed later. The dip in amplitude at $\pm$ 5 Kc is a resonance coupling artifact.

Verification of Statement 3—that major changes in the dynamics of cochlear microphonics are due almost exclusively to middle ear muscle activity—is provided by ten such completely demuscled cats. In none of these was any significant difference observed between the awake and the anesthetized recordings.

Thus, having established that any major differences between awake cochlear microphonic measurements and anesthetized ones are caused by the tensor and stapedius, the same experiments were carried

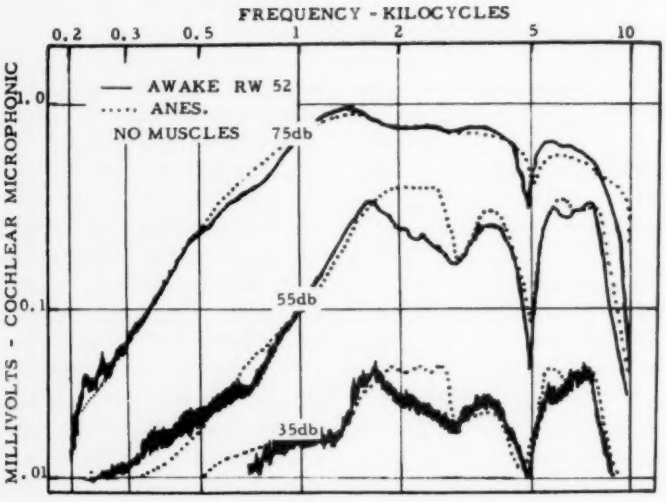



Fig. 1.—Round window cochlear microphonic responses to 3 intensities of a swept-frequency tone stimulus. These are awake (solid lines) and anesthetized (dotted lines) recordings from a cat in which both middle ear muscles have been cut. Indicated intensities are for 1 Kc, relative to decibels above human threshold.

out on cats with intact middle ear muscles. Figure 2 illustrates one such experiment identical in protocol with the Figure 1 demuscled ear. As before, the solid lines are measurements of microphonic in the awake animal, and the dotted lines are the anesthetized levels. It is apparent from this figure that the middle ear muscles exert considerable influence on cochlear microphonic even at low intensities, such as 35 db.

Though this particular animal showed more microphonic attenuation than most, others had even larger awake-anesthesia differences. One animal's middle ear muscle activity reduced the cochlear microphonic 30 db at 700 cps in response to this same 75 db tone. On the other end of the attenuation continuum were two cats showing no evidence of contraction, and a few producing only 4-5 db of reduction.

Another and more universal feature of middle ear muscle action demonstrated by Figure 2 is the greater response at lower frequencies.

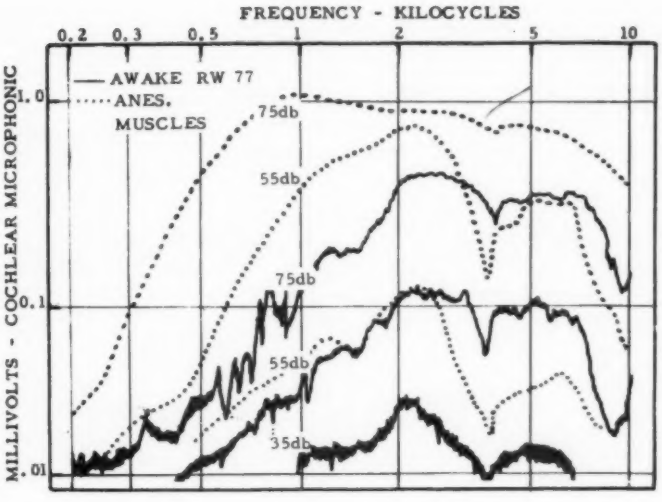



Fig. 2.—Round window cochlear microphonic responses to 3 intensities of a swept-frequency tone stimulus. These are awake (solid lines) and anesthetized (dotted lines) recordings from a cat with the middle ear muscles intact. Stimulus parameters are identical to those of Figure 1.

In each cat, no matter what the over-all level of attenuation, there was always considerably more effect below about 2 Kc.

This low-frequency preference and the range of middle ear muscle contractions are more appropriately demonstrated through summarized data of all the normally muscled cats, Figures 3 and 4. Though these two figures are structurally different, each was derived from the same basic data on 32 ears of 25 cats. A more comprehensive understanding of middle ear muscle activity can be gained when the two figures are treated as a unit.

Figure 3 is constructed so as to compare the relative cochlear microphonic amplitude changes observed in the awake cat with those observed in the same animals anesthetized. In some respects it is simply a refinement of the raw data exemplified by Figure 2. The microphonic amplitude of the awake animal has been made the baseline value (ordinate zero). Anesthetized levels are recorded in decibels of change from the awake levels. In the interest of thoroughness, data from the control (demuscled) cats are plotted in the same man-

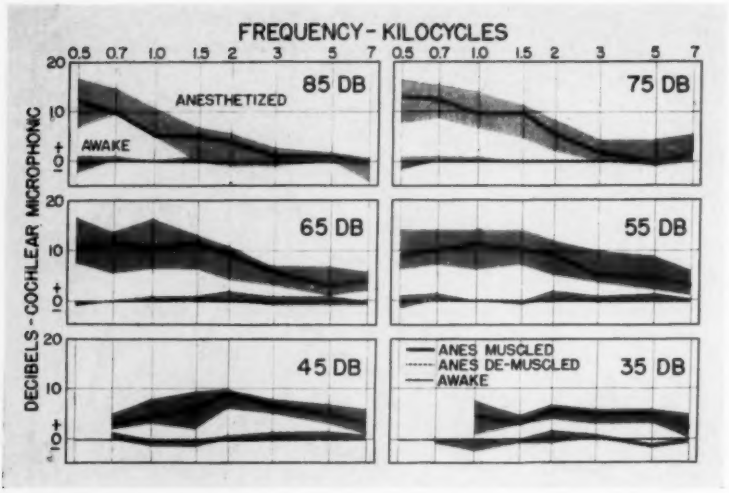



Fig. 3.—Relative differences in cochlear microphonic amplitude of awake and anesthetized cats. The difference in decibels—between microphonic amplitudes of the awake cat (ordinate zero) and the same animals anesthetized—are plotted for 32 measurements on 25 muscled ears (heavy solid line) and 10 de-muscled ears (dashed line). These are median values. Shaded areas indicate interquartile range. Indicated sound intensities are relative to human threshold at 1 Kc.

ner. For example, the *median* attenuation of the normally muscled ears for a 1 Kc tone at 75 db above threshold was + 10 db (heavy solid line). For the demuscled animals the awake-anesthesia difference was zero db (dotted line). The shaded areas indicate interquartile ranges of both groups.*

These same changes are plotted in absolute values of cochlear microphonic on the ordinate of Figure 4. Ten-db increments in sound level are indicated along the abscissa from left to right. As before, solid lines are awake measurements, and dashed ones anesthetized. Thus the same 1 Kc tone exemplified in Figure 3 can now be read in terms of microphonic isopotential. For example, sound intensity

*Mean and std. dev. were also calculated. For the control group means were less than 1 db with std. dev. usually less than 1 db. For the muscled group, 3 db was the max. deviation of mean from median. Standard deviation also corresponded with the interquartile range, but failed to distinguish skewness, especially at higher frequencies.

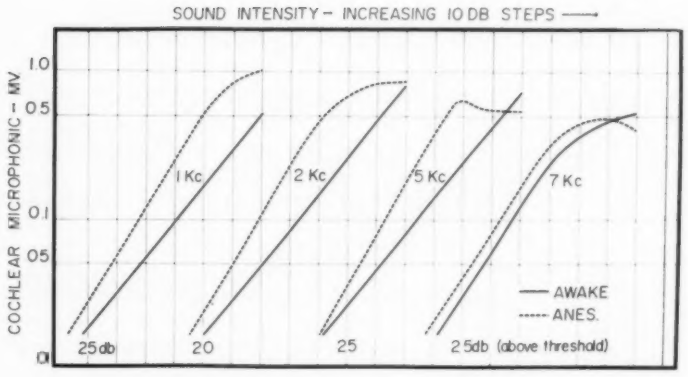



Fig. 4.—Awake—anesthetized—cochlear microphonic frequency response curves of 25 normally muscled ears. (Mean values). Ordinate divisions are 10 db increments in sound intensity relative to human threshold.

must be increased 20 db in an awake normally muscled ear to produce the same cochlear microphonic response as that generated in this same ear anesthetized.

As can be seen by either method of presenting the data, the tensor and stapedius cause a considerable reduction in the cochlear microphonic response of the awake animal. Furthermore, this attenuation is not the exclusive property of loud sounds. Quite significant reductions are evident for tones of moderate or even low intensity. In general the more intense sounds cause more muscle contraction. This is in contrast to what might be inferred from a hurried glance at Figure 3, where the relative attenuation seems less at 85 db than it does at 65 db above threshold. The discrepancy can be clarified by examining the absolute values of Figure 4. The loss of middle ear muscle contractions in the anesthetized animal allows much more intense sounds to reach the cochlea. At higher sound levels, this increased energy is enough to introduce non-linear distortion in the cochlear microphonic. Thus the relatively smaller microphonic levels result not from decrease, but from over-stimulation.

Further consideration of attenuation of moderate sounds by the tensor and stapedius cannot be divorced from considerations of frequency. As has already been mentioned, and as can be seen in the two figures, especially Figure 3, the lower-frequency sounds are rela-

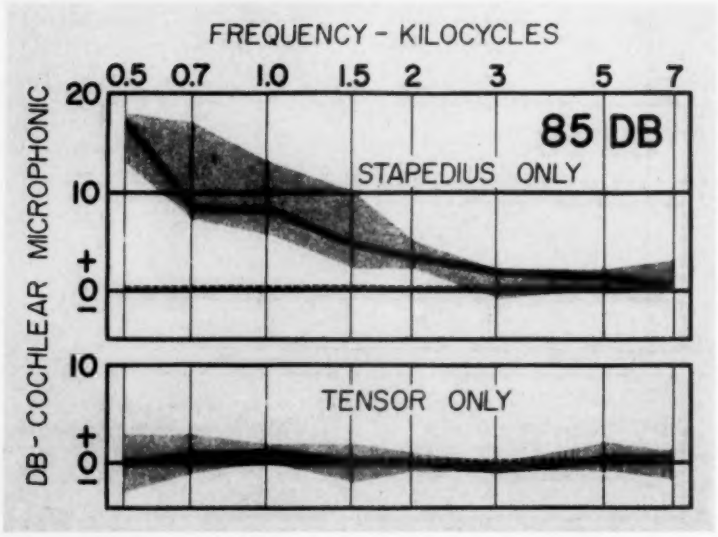



Fig. 5.—Relative difference in cochlear microphonic amplitudes of awake and anesthetized cats with only one middle ear muscle intact. The difference (in decibels) between microphonic voltage of the awake cat (solid line) and the same animals anesthetized (dashed line) are plotted for median values of 25 ears. Shaded areas indicate interquartile range. Sound level is 85 db above human threshold at 1 Kc.

tively much more affected. In general, greater microphonic attenuation is registered for tones below about 2 Kc. Above this point, middle ear muscle activity decreases gradually until, at 7 Kc, there is relatively little difference between the cochlear microphonic of an awake, active middle ear muscled cat and one that is anesthetized or demuscled. Though the gradually changing aspect of frequency is de-emphasized somewhat by the plotting of a mean value in Figure 4, the contrast between the effects of identically intense (in terms of threshold) 1-Kc and 7-Kc tones is self-evident. More will be said about these factors later. For now, it is sufficient to say that in cats with both the stapedius and the tensor intact, large reductions of cochlear microphonic occur, particularly at lower frequencies.

*Tensor or Stapedius.* In order to clarify the individual function of the middle ear muscles, 25 cats were prepared with only one of the tympanic muscles left intact. Most of these animals were bilateral

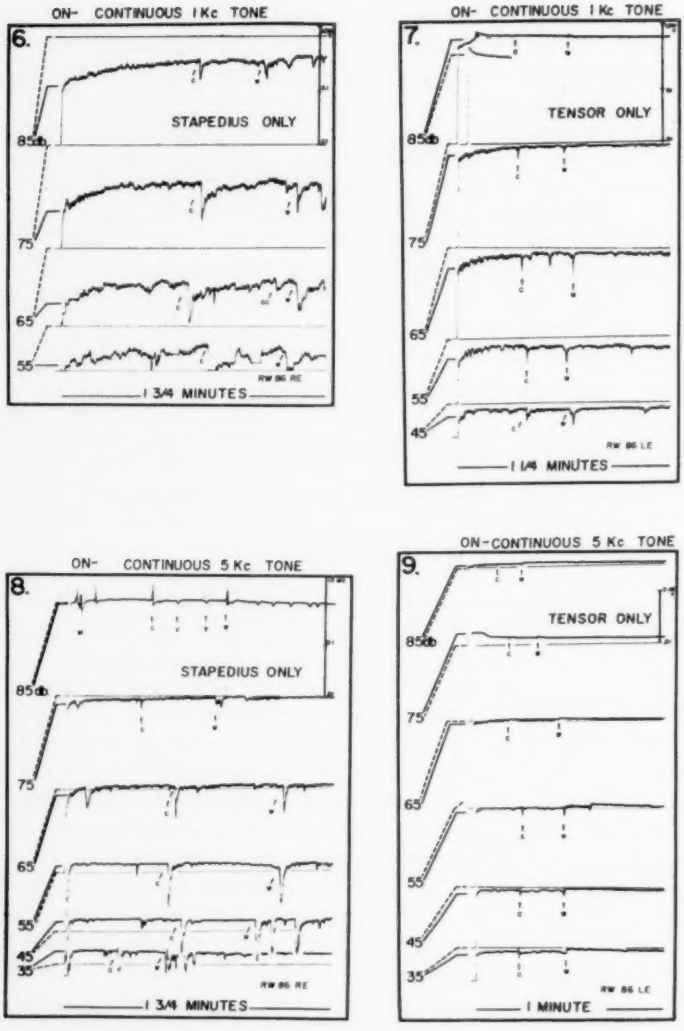
implantations with the tensor tympani cut in one ear and the stapedius cut in the other, but a few of each group had both muscles left intact in the opposite ear. These cats were tested under the same circumstances as the animals already described. The comparative activities of the two muscles at the 85 db level appear in Figure 5. This figure is similar in construction to Figure 3, except that the dotted line here represents anesthetized level (zero) and not a completely demuscled ear. The other sound-intensity levels (35-75 db above threshold) were also much like those of Figure 5.

Although some evidence of small amounts of microphonic attenuation was found in two or three of the cats prepared with only the tensor intact, Figure 5 does not exaggerate the relative insignificance of its role in the reduction of cochlear microphonic in the awake cat.* In most instances tensor-only recordings were difficult to distinguish from demuscled ones.

On the other hand, separation of stapedius-only recordings from those of fully-muscled ears was impossible without reference to the surgical protocol. Thus, it is permissible to say that most of the dynamic action of the middle ear muscles in response to moderate sound can be credited to the stapedius.

Another demonstration of the difference in activity of the two muscles is seen in Figures 6-9 which compare tensor and stapedius influences on the cochlear microphonic in response to a continuous, single-frequency tone instead of a swept one. Figures 6 and 8 are from the right ear of a cat that has only the stapedius intact. Figures 7 and 9 are recorded from the left ear of this same cat in which only the tensor remains. The top two records are for responses to a 1 Kc tone, and the lower two for a 5 Kc one. In each instance the baseline (no tone) level of cochlear microphonic has been adjusted to read 10 uv. Anesthesia levels for the same stimulus have been drawn in for comparison (dashed lines). The awake response begins with the first upward deflection of the ink-writer on the left-hand side of each figure. This tone remains on, without interruption, for the times indicated below each figure.

*This relative inactivity of the tensor tympani for moderate-intensity sounds may well explain the discrepancy between the reflex activity reported here and the human thresholds reported by Jepsen.[5] Jepsen, using Metz's acoustical impedance bridge methods, measured changes in the position of the tympanic membrane in response to contralateral sound. Presumably, variations in the position of the tympanic membrane reflected contraction and relaxation of the middle ear muscles. While it is conceivable that such measures might give a rough estimation of tensor activity, it is difficult to see how stapedius contractions would affect the tympanic membrane.




Figs. 6 - 9.—Cochlear microphonic amplitude responses to continuous tone stimulus. In the left-hand figures, the stapedius is intact; right-hand figures are from a tensor tympani-only ear. Sound levels are relative to human threshold for both the awake (solid lines) and the anesthetized (dashed lines) microphonic levels. Tone turned on at left of each recording. C—hand clap, W—wisker flick, V—vocalization, M—movement artifact.

To illustrate further some aspects of middle ear muscle contractions, two additional contraction-producing stimuli were introduced part way through the tone presentation. "C" refers to a single hand clap, produced by the experimenter from about two feet away from the cat. The other stimulus "W" was non-auditory, produced simply by flicking the tips of the cat's contralateral whiskers with the end of a pencil. It can easily be seen that the stapedius response is more active in reducing cochlear microphonic level with either stimulus.

Close inspection of the hand clap response can also give the reader some information regarding the relative reflex latency of the two muscles. The microphonic response to hand clap in the tensor-only ear is initially upward. In the stapedius-only ear it is usually downward. The early upward movement was caused by cochlear microphonic response to the clap. In the stapedius-only ear, muscle contraction was early enough so that, in combination with the relatively slow pen movement, the hand clap microphonic did not register.

More will be said about these figures shortly. For now, it is enough to demonstrate that the stapedius is far more active than the tensor under these circumstances, and that the only apparent difference in their effects on cochlear microphonic amplitude is one of degree.

*Stimulus Parameters.* It is reasonable to assume that some types of sounds will be more likely to cause reflex contractions than others. Unfortunately, it is difficult to compartmentalize stimulus parameters so as to be able to deal with each one individually and dismiss it from consideration of the others. The three most important factors in production of middle ear muscle contractions appear to be frequency, intensity, and change of stimulus (adaptation). For example, the ideal stimulus for middle ear muscle contraction would be a loud, randomly intermittent sound composed of constantly changing, low-frequency tones.

Though chosen more or less arbitrarily at the beginning, the swept-frequency stimulus used in part of these experiments fulfilled some of these criteria rather well. In a given animal such as RW 86 (Figures 6-9) this frequency-modulated tone almost universally produced more sustained middle ear muscle contraction at all frequencies and intensities than did a single continuous tone. For instance, the cochlear microphonic response to the 1 Kc tone (Figure 6) at 55 db above threshold did not even appear above the 10 uv baseline in the

swept-tone measurements. This difference is, of course, due to an adaptation phenomenon, which in turn is closely related to frequency.

*Adaptation.—Frequency.* Middle ear muscle contractions are capable of reducing cochlear microphonic amplitude at any frequency, and when stimulated with sudden short sounds they do just this. When the stimulating sound continues over longer periods of time, the attenuation of high frequency sounds (4-5 Kc and above) suffers from adaptation much more rapidly than do the lower ones. Note the differences in relaxation time after onset of the 1 Kc tone and the 5 Kc one (Figs. 6 and 8). This preferential treatment of low frequencies and their adaptation time is not just a simple matter of "reflex fatigue." There seem to be more subtle points involved. Note the muscle response to a hand clap at the lower intensity levels where masking is least likely to be involved. This hand clap had a duration of much less than a second. Yet the muscle responses to it were as if sound had been introduced for the first time. This sudden change of stimulus was enough to reset the adaptation cycle so completely that it closely resembled the original produced when the pure tone was first presented.

The slope of the muscle relaxation curve (adaptation) is closely related to frequency. In general, the lower the frequency, the more gradual the adaptation. This relationship holds within each individual cat, but actual values of microphonic reduction and relaxation time vary considerably among cats.

*Adaptation.—Intensity.* The role of sound intensity in adaptation is not entirely clear from these experiments. One reason for this, namely non-linear distortion of the cochlear microphonic, can be seen in Figures 4 and 8. Follow what happens in the muscle response to hand clap as sound intensity increases. The cochlear microphonic reduction gets smaller as intensity gets larger, until, at 85 db above threshold, the muscle contraction apparently *increases* the cochlear microphonic amplitude. Note the same pattern for the non-auditory stimulus. Though real, this increase is nothing more than the reduction of distortion.

As a consequence, the actual amount of microphonic reduction produced by equal amounts of muscle contraction is also a function of where on the intensity-amplitude curve the contraction occurs. However, it is the experimenter's impression that the importance of sound intensity in adaptation is of lesser magnitude for low intensities, and gradually assumes more importance as intensity increases. For instance, a 10-second, 1 Kc tone at 50 db above threshold would be

more effective than a 70 db one at 5 Kc, but a 70 db 1 Kc tone would have a longer adaptation time than either of these.

*Reflex Threshold.* As is the case with hearing itself, there does not appear to be an absolute acoustic reflex threshold, but rather a somewhat labile one, dependent on many factors. The variance of muscle activity is reflected in the wide interquartile ranges of Figure 3. Even individuals within this group had large shifts in muscle response from one day to the next. Similar evidence of varying thresholds has been obtained from another group of awake cats which had electrodes implanted directly in the tensor and stapedius. Absolute or variable, the reflex threshold does extend through the range of moderate-intensity sound.

*Static Effects.* The middle ear muscles undoubtedly do influence ossicular transmission even when they are not contracting. For this particular experimental situation, the two most important factors are 1) the additional mass and 2) the support provided to the ossicular chain by the normal tonic muscles of the awake cat, as contrasted with the reduced muscle tone of the anesthetized cat. For the most part, the pertinent evidence that the above-described experiments can provide is indirect and inconclusive.

Within a rough approximation, passive muscle mass has little influence on threshold transmission or on maximum values of cochlear microphonic. Cats with both muscles cut generated amplitude and frequency contours of cochlear microphonic similar to those of normally muscled ears under anesthesia.

The second factor, muscle tonus and ossicular support, is more difficult to assess. Where does one draw the line between normal muscle tone and active contraction? For a normally muscled ear of this series it cannot be drawn, since awake-anesthesia differences ranged from zero to 30 db. If muscle tone were a constant factor, one would expect some standard amount of elevation or depression of cochlear microphonic with anesthesia. In the group of cats with one of the two muscles cut, the minimal changes in the measurements of tensor-only cats virtually eliminate this muscle from any more than token participation in these effects. This same conclusion was reached by Wever, et al.[6] The situation for stapedius-only cats is another matter, comparable in nature to that of the normally muscled ears.

One can infer from the studies of Wever et al. that actual ossicular displacements (presumably a function of isotonic contraction) are

only one important factor. Sound attenuation by damping with little ossicular displacement (isotonic contraction) and normal muscle tonus must also be considered. Though this question must be left open until more definitive experiments can be done, it is the experimenter's impression that normal muscle tonus did not contribute more than 5 db of attenuation to the above-mentioned awake-anesthetized differences.

The muscular support of the ossicular chain does alter slightly the resonant frequency of the conduction mechanism. Normally muscled awake animals had resonant points about one-fourth to one-fifth octave higher than when they were anesthetized. This was not observed in demuscled ears.

The other static change in cochlear microphonic with anesthesia appears in Figure 1. This elevation between 1.7 Kc and 2.7 Kc appeared in muscled and demuscled ears alike, but not universally in either group. The most likely explanation for this is another resonance shift, possibly due to middle ear volume changes, through anesthetic effects on eustachian tube structures.

## SPECULATIONS

It is hoped that by this time the reader is at least partially convinced that the activities of the middle ear muscles are important at moderate sound levels in the cat. If one is willing to equate humans with cats for purposes of discussion, a few further points may be worth speculation.

*Psychoacoustics.* The extension of the range of middle ear muscle contractions may have implications for some psychoacoustic experiments. For example, it is entirely possible that certain masking effects and perstimulatory fatigue phenomena may be wholly or partly the result of middle ear muscle attenuation. Furthermore it is conceivable that spontaneous contractions, or those responding to incidental stimuli of a poor testing environment, may be a partial explanation for apparent instability of threshold measures in some subjects. Some evidence, gathered under such poor control conditions that it was not described in "Results" from the experiments herein reported, suggests this.

When recording cochlear microphonics in the awake cat, the author had to be rather careful not to introduce incidental sounds, since even loud whispers in the immediate vicinity of the cat, or noisy

talk outside the door of the testing booth, would produce muscle contractions when the sound level being presented to the cat was low. One fortuitous and rather crude example of this appears in Figure 6 at a higher sound level (65 db). Note the response marked "G.C." This contraction occurred in response to the sound of a garbage can lid dropped on the sidewalk somewhere outside the building.

*Acoustic Trauma.* This author is in accord with Smith and Wever[3] when they say: ". . . It is fortunate that the greater protection (of the middle ear muscles) is afforded for the low tones, because the stimulation deafness experiments show that when tones are of equal pressure their destructiveness increases as the frequency becomes lower." From this standpoint, little more need be said. However, the problem of individual susceptibility to acoustic trauma remains unsolved. It is reasonable to assume that if as much individual difference in middle ear muscles activity exists in humans as it does in cats, one explanation of this susceptibility is at hand.

*Auditory Physiology.* Admittedly, the response of the stapedius and tensor tympani muscles is only a reflex. But, as Lorente de No[7] emphasized, it has the potential of being an extremely powerful tool for studies of auditory physiology. His reasoning becomes more lucid if one is willing to concede that the motor nuclei involved* are but intermediates reflecting the efferent input of the auditory system directly and the reticular core indirectly. This is exemplified even in the preliminary experiments described above, where a brief additional stimulus such as a hand clap was enough to reset almost completely a frequency-specific adaptation cycle. This performance, when interpreted in the light of current cochlear theory, is substantial electrophysiological evidence for central adaptation to both frequency and intensity.

Portions of this concept are not completely new. Kobrak,[8] working on the tensor tympani reflex in rabbits, reported fatigue phenomena, most of which, he felt, were cochlear in origin, but not all. Possibly his conclusions were colored by the high sound levels used and the longer intervals of time that he measured. Using the impedance method on humans, Metz[3] reported that reflex fatigue was abolished by interrupting the sound stimulus only briefly.

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*The connections of the auditory efferents to the V and VII motor nuclei are not well worked out. In the cat there is some evidence for a superior olivary connection.[7] Whatever the pathway, it probably does not involve many synapses since the minimum reflex latency from onset of cochlear microphonic to stapedius EMG potentials is about 6 msec.

There is further evidence for the interaction of reticular and auditory systems at brain stem levels in the work of Sharpless and Jasper[9] who studied habituation of cortical arousal in response to repeated tones. Though their work was on the higher nervous system, they too were impressed by the effects of disinhibition produced by novel stimuli such as frequency shift.

Disinhibition phenomena themselves are no newer than other Pavlovian concepts such as inhibition, habituation, etc. Nor is frequency specificity in itself enigmatic when one stops to think about it. The important factor is the happy circumstance of an easily observable reflex arc, intimately associated with the auditory system and influenced by brain stem reticular activity.

## SUMMARY

The effects of the middle ear muscles on cochlear microphonic were studied in awake and anesthetized cats by means of electrodes permanently implanted on their round windows. Continuous single- and swept-frequency tones were used to study frequencies between 0.2 and 7 Kc at intensities from 20-25 db to 85-90 db above human threshold. Evidence is presented that:

1. Dynamic changes in the cochlear microphonic of a normal awake cat are due almost exclusively to the action of the middle ear muscles.

2. Middle ear muscle contractions produce significant attenuation in the transmission of moderate-intensity sounds across the middle ear. The over-all effect of this activity reduces distortion of the cochlear microphonic and increases its linear range, especially for the lower frequencies.

3. The tympanic muscle acoustic reflex can be activated by any tone, but the resultant contractions adapt more quickly and are generally less effective at higher frequencies.

4. The characteristics of this frequency-specific adaptation cycle can be interpreted as further evidence of relatively rapid central-nervous-system adaptation to both frequency and intensity at the medullo-pontine level.

5. The stapedius is primarily responsible for the above-listed effects.

6. Considerable variation in middle ear muscle activity is to be found from cat to cat.

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LXXX

# CHONDROMA OF THE LARYNX

## REPORT OF A CASE AND A REVIEW OF THE LITERATURE

WILLIAM L. WALTER, M.D.

STATEN ISLAND, N. Y.

A.M., a 58 year old merchant seaman, was admitted to the United States Public Health Service Hospital, Staten Island, New York, on November 25, 1957, with a history of gradual progressive hoarseness of three months' duration. This hoarseness was of insidious onset, and was not preceded by an upper respiratory infection. The history also revealed an asymptomatic left neck mass of two weeks' duration.

Physical examination revealed a well-developed, well-nourished, white man in apparent good physical condition. His voice was hoarse, but he showed no evidence of any respiratory difficulty. Breathing was normal. A firm, nodular, slightly mobile, and non-tender mass, which shifted upward whenever the patient swallowed, was palpable on the left side of the neck in the general area of the left lobe of the thyroid gland. No definite attachment of this mass to the outer portion of the larynx was discernible upon physical examination. Direct laryngoscopy revealed the entire larynx shifted to the right by the left neck mass, and a complete paralysis of the left vocal cord was present; the supra and subglottic laryngeal areas showed no evidence of any deformity, and no intralaryngeal lesions were visible. The clinical impression at this time was an enlargement of the left lobe of the thyroid gland with pressure upon the left recurrent laryngeal nerve, causing a left vocal cord paralysis. A clinical suspicion of a thyroid malignancy was entertained.

An x-ray of the neck revealed a very fine mottled area of calcification appearing to be in the general region of the left lobe of the thyroid gland (Fig. 1). The radiologist thought this might represent

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either a Riedel's struma or calcification within a thyroid carcinoma of low grade malignancy. The possibility of some type of calcifying tumor was also considered.

Extensive laboratory tests, including thyroid function studies (B.M.R., R.A.I., and other blood examinations), were all within normal limits. Tomograms of the larynx and barium swallow studies were not done.

The neck mass was considered to be either a calcified thyroid adenoma or perhaps some type of a calcifying tumor. An operation was done on December 20, 1957, and surgery revealed a mass on the left side of the neck located beneath the left lobe of the thyroid gland and displacing the normal appearing left thyroid lobe anteriorly. Upon further exposure, this mass was found to originate from the left posterior aspect of the cricoid cartilage. The tumor mass was found to be causing pressure upon the left recurrent laryngeal nerve. The growth was firm and calcified in density, and measured approximately one and one-half inches in diameter. It did not involve the thyroid gland, esophagus, or pharynx. There was a partial cleavage plane existing between the mass and the thyroid gland, esophagus, and some of the adjoining soft tissues, and this permitted easy separation of the tumor from these surrounding structures. However, there was no cleavage plane between the cricoid cartilage and the tumor itself. On the contrary, because of local extension of the growth, the origin of the tumor was found to be firmly attached by a very broad, firm, and adherent base which not only included the cricoid cartilage, but also involved laryngeal and neighboring soft tissues, including portions of the posterior cricoarytenoid, cricopharyngeus, and cricothyroid muscles (Fig. 2). No palpable regional lymph nodes were present. A frozen section proved the lesion to be a cartilaginous tumor, either a chondroma or possibly a chondrosarcoma. Because of the broad, firm attachment of the base of the growth to the cricoid cartilage and the bordering laryngeal tissues, a considerable portion of the larynx would have had to be sacrificed in order to remove the tumor in its entirety. This would most probably result in collapse or stenosis or both, of the larynx, so it was decided that a total laryngectomy was the procedure of choice.

The left lobe of the thyroid was removed. The thyroid cartilage, cricoid cartilage with the attached mass, and the upper portion of the trachea were mobilized. Section of the trachea was done below the third cartilaginous ring. The upper portion of the trachea and larynx were then dissected up posteriorly with all vessels carefully

Fig. 1.—X-ray showing the lateral aspect of the neck. The arrows indicate irregular mottled areas of calcification within the tumor mass.

ligated. The epiglottis, larynx with the attached tumor, and the upper three tracheal cartilaginous rings were then removed. The pharyngeal defect was approximated by a "T" type of closure after a levine tube was passed into the stomach under direct vision. The tracheal ostia was sutured to the skin; a tracheostomy tube was inserted, and the wound closed without any drains. The procedure was tolerated very well by the patient, and he was taken to the recovery room in good condition.

The thyroid gland and laryngeal specimens were submitted to the pathology department for pathologic diagnosis. The pathologist examined the tissues and gave the following report: The examination of the thyroid tissue submitted shows no evidence of any pathologic changes. The entire larynx and cartilages surrounding it are submitted together with the first three tracheal rings. Attached to the

left lateral posterior portion of the cricoid cartilage is a very firm elliptical shaped gray mass measuring 5 cm x 4 cm x 3.5 cm. The external surface is smooth, grayish in color, and very firm in consistency. The base where it is attached to the cricoid cartilage measures 4 cm x 4 cm. The cut surface is gray with a ground glass appearance. The center of the lesion reveals cystic degeneration. Microscopic examination of the tumor shows nodular very cellular hyaline cartilage with a very disorganized pattern. The nuclear pattern is irregular with pleomorphic, vesicular, hyperchromatic nuclei (Fig. 3).

Diagnosis: Chondroma of the cricoid cartilage.

Comment: Microscopic examination of some sections of the tumor suggested that the lesion might possibly be a chondrosarcoma of very low grade. Sections of the tumor mass were submitted to the Armed Forces Institute of Pathology, and the diagnosis of chondroma of the larynx was confirmed.

The patient's first five postoperative days were uneventful with the patient fully ambulant by the second postoperative day with no complications. On the sixth postoperative day, approximately 30 minutes after receiving his routine A.M. levine tube feeding, the patient developed a sudden, severe, sharp, stabbing pain in his left chest over the precordia area. He rapidly went into shock with dyspnea and cyanosis, and was dead within five minutes of the initial attack. The tracheal stoma was found to be patent and unobstructed. Unfortunately, an autopsy was not permitted in this case. The cause of death was thought to be due to an acute myocardial occlusion.

## DISCUSSION OF THE LITERATURE

Willis[1] considers chondromas as benign lesions belonging to the group of solitary well-circumscribed cartilaginous tumors of the larynx or other parts which grow slowly and non-invasively, and for which a good prognosis can be given following adequate removal.

These cartilaginous tumors are rare in the larynx as compared with their incidence in other regions of the body.[2] Chondromas of the laryngeal cartilages are only occasionally encountered by the laryngologist. This is very much in evidence when reported cases of these tumors are searched for in the literature. Of course, it is known that the literature of any particular subject represents only the number of cases reported; however, the infrequent occurrence of these

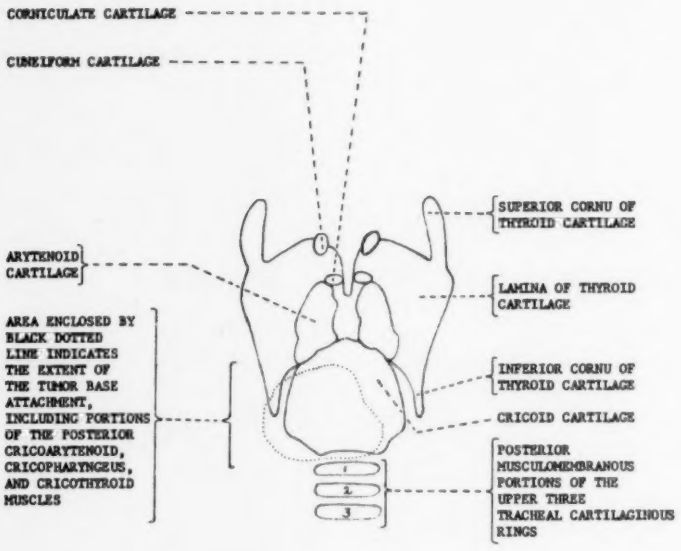
CORNICULATE CARTILAGE
CUNEIFORM CARTILAGE
ARYTENOID CARTILAGE
AREA ENCLOSED BY BLACK DOTTED LINE INDICATES THE EXTENT OF THE TUMOR BASE ATTACHMENT, INCLUDING PORTIONS OF THE POSTERIOR CRICOARYTENOID, CRICOPHARYNGEUS, AND CRICOTHYROID MUSCLES
SUPERIOR CORNU OF THYROID CARTILAGE
LAMINA OF THYROID CARTILAGE
INFERIOR CORNU OF THYROID CARTILAGE
CRICOID CARTILAGE
POSTERIOR MUSCULOMEMBRANOUS PORTIONS OF THE UPPER THREE TRACHEAL CARTILAGINOUS RINGS
1
2
3

Fig. 2.—A skeletal outline of the laryngeal cartilages as viewed from behind. The posterior musculo-membranous portions of the upper three tracheal rings are included. The area enclosed within the black dotted line indicates the extent of the base attachment of the chondromatous tumor.

particular laryngeal tumors has been substantiated by the clinical experiences of older investigators.

Heusinger[3] was the first person to give a description of chondroma of the larynx. Moore[4] surveyed the world's literature and collected 53 cases of true laryngeal chondroma. McCall, Dupertius, and Gardiner[5] reported two cases of their own, and were able to collect an additional 30 cases since Moore's earlier report. Rosedale,[6] Moore,[7] Link[8] and Ryan and Zizmor[9] reported single cases. Huet and Labayle[10] each described a case, and Frenzel[11] reported three additional cases of laryngeal chondroma. Mattiolo[12] reported a case of giant mixed chondroma ossificans of the larynx, and Dominik[13] added another case of laryngeal chondroma to the literature. Therefore, 96 cases of chondroma of the larynx have been reported in the literature. The case presented here constitutes the 97th reported case. Foxwell[14] reported a case of solitary chondroma of the trachea.

Because of the fact of its unusual occurrence, I believe the presentation of this case of laryngeal chondroma which I have observed is justified. The purpose of this paper is to add another case of this condition to the literature and to discuss various aspects of chondroma of the larynx.

## PATHOLOGICAL ASPECTS

Chondromas are neoplasms of mesodermal origin, arising out of and showing definite histologic characteristics of various types of cartilage, mainly the hyaline variety. Although certain areas of the matrix found in these tumors may have become myxomatous, calcified, or ossified, the basic proliferating tissue will be found to be cartilage. These tumors can be found occurring throughout the general skeletal system. They grow quite slowly and often attain large sizes. Sometimes tumors of relative large size are found in areas of poor blood supply. This is because cartilage is lymph nourished, allowing chondromas to develop to a relative good size in sites which normally would be thought of as having a precarious blood supply. As a result of a slowly expanding tumor, there may be some pressure atrophy of the adjoining tissues; therefore, when the larynx is involved, it may not be possible to remove the tumor without harming the thinner structures surrounding or lining this organ.

Ewing[15] has stated that localized overgrowth of cartilage occurs in several forms between which it sometimes becomes difficult to draw clear distinctions. Limited outgrowths of pre-existing cartilage occur on the ribs, in the larynx, and about joints which exhibit the characters of a simple hyperplastic process and are called ecchondroses. True progressive neoplasms composed of cartilage appear in the same situations and also in tissues not normally containing cartilage and these are called chondromas or ecchondromas. That many ecchondroses possess some of the properties of tumors is indicated by the occasional occurrence of large chondromas as a sequel of ecchondroses.

Clinically, this differentiation is important because chondromas, which spring from any of the laryngeal cartilages, usually destroy the larynx by involving and obliterating the normal laryngeal framework. On the other hand, those tumors originating in the tissues in the region of the larynx, completely disconnected from the laryngeal cartilages, may compress the larynx almost to the point of obliteration. These latter tumors can usually be completely "shelled out," and when once removed, the uninvolved laryngeal cartilages can resume their original shape, and the laryngeal function is resumed. Unfortunately, this

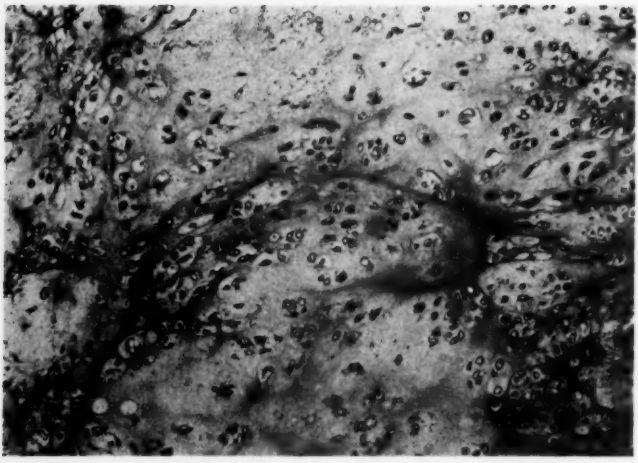

Fig. 3.—Photomicrograph showing cellular hyaline cartilage with a very disorganized pattern. The nuclei are pleomorphic, vesicular, and hyperchromatic with an irregular pattern (H. & E. x 80).

type of tumor arises less frequently. Most chondromas arise as a localized overgrowth from laryngeal cartilage, and according to Rosedale,[6] if the tumor originates from an anatomic cartilaginous area, it does not have a true capsule in relation to its origin. The expanding growth pushes aside or forward any surrounding soft tissues, and in doing so, a partial cleavage plane is developed. However, this plane of cleavage does not exist between the origin of the tumor and the tumor itself. Therefore, these tumors have to be removed with the scalpel or by morcellation, as they cannot be "shelled out."

In regard to the site of location of laryngeal chondromas, Link[8] found that according to the reviews of all reported cases prior to 1949 the favorite site of development of chondroma of the larynx was on the endolaryngeal surface of the posterior plate of the cricoid cartilage, followed by the thyroid cartilage, the epiglottis, and the arytenoid cartilage in order of frequency, with the greatest number arising from the cricoid cartilage. In the case presented in this paper, the lesion originated from the posterior aspect of the cricoid cartilage.

Chondromas usually form as solid, hard, lobulated, rounded tumors which may attain large dimensions.[15] Most of the tumors are

solid, but many become softened by mucinous degeneration, and cysts, filled with serous, mucinous, or fatty material may form.

Chondromas consist of the same type of cartilage from which they originate, but sometimes in an altered form.[5] They are composed of well-differentiated inactive-looking cartilage, either of healthy appearance or mucoid and soft. Some tumors show calcification or ossification or both. The cartilage cells are usually irregularly dispersed in a matrix of hyaline or fibroelastic substance, and may be more or less numerous than in normal cartilage; the cells themselves are characterized by vacuolated or clear staining stellate or ovoid nuclei.[8]

In their discussion of the microscopic pathology of chondromas, Lichtenstein and Jaffe[16] remarked that the cells making up these tumors showed definite characteristics such as 1) the component cartilage cells are fairly consistently rather small; 2) the vast majority of them have only one nucleus; 3) this nucleus tends strongly to be rather small and definitely not plump in relation to the cell as a whole; 4) such cells as are binuclear usually are still small, still have small (and also not plump) nuclei, and are found only occasionally and even then in scattered fields; and 5) in particular there are no large or giant cartilage cells with large single or multiple nuclei or with clumps of chromatin.

Chondromas do not metastasize, but have a marked tendency to recur locally, sometimes in large bulk, after surgical intervention, if any part of the growth remains. McCall, Dupertius, and Gardiner[5] reported a case which showed recurrence of the tumor after previous surgical techniques failed to completely remove all of the lesion. The recurrent mass was so extensive that it finally became necessary to perform a total laryngectomy, with removal of the upper five tracheal rings, anterior wall of the esophagus, and thyroid gland in order to completely remove all of the growth.

## CLINICAL ASPECTS

*Etiology.* To my knowledge, the specific cause of these particular cartilaginous neoplasms of the larynx has so far been undetermined, although numerous theories have been advanced to explain their development. Certain factors which seem to be involved are age, sex, and ossification. Chondromas of the larynx are usually thought of as diseases affecting adults, mainly between 40 and 60 years of age. Of the reported cases of laryngeal chondroma, men were found to be

more affected than women, the ratio being approximately 10 : 1. Taylor[17] showed that ossification of the laryngeal cartilages begins later and progresses slower in women than in men. This may be the reason why the majority of these tumors arise in males.

*Symptoms.* The symptoms resulting from these lesions will vary depending upon the tumor's location, size, and rate of growth. Usually these growths develop quite slowly, and their presence may be detected only after they begin to interfere with normal function. Tumors which develop from the outer walls of the thyroid cartilage or outside the larynx, unless they involve the recurrent laryngeal nerve, may cause no symptoms other than a complaint of swelling in the neck. Such tumors will remain relatively asymptomatic until encroachment upon the interior of the larynx is present.

Intralaryngeal growths may cause mechanical obstruction of the movement of one or both vocal cords. If laryngeal motility is interfered with, hoarseness may be a common, early, and perhaps the only symptom for some time. In later stages, whispered voice and aphonia will be present. In the case presented in this paper, hoarseness resulted because of left vocal cord paralysis due to pressure of the tumor upon the left recurrent laryngeal nerve. A lesion which involves the inner aspect of the thyroid cartilage may give rise to early hoarseness, but similar lesions involving the epiglottis, cricoid plate, and arytenoid cartilages rarely cause hoarseness alone.

Wheezing, dyspnea, and finally asphyxia result when the lesion arises from the inner aspect of the posterior plate of the cricoid cartilage and encroaches upon the breathing space. According to Havens and Parkhill,[18] dyspnea and sometimes stridor can develop quickly in previously asymptomatic individuals because of swelling of a subglottic tumor due to an inflammatory reaction or sudden increase in the rate of growth of the lesion.

Sometimes, owing to the increasing bulk of tumors situated upon the posterior surface of the cricoid cartilage, dysphagia may develop because of pressure upon the esophagus. This is usually a late symptom however.

Pain may be present with large extrinsic growths and may be accentuated by swallowing.

Cough is present occasionally and may be the most troublesome symptom present.

*Physical Findings.* These will also vary depending upon the size and location of the lesion. If the tumor has extruded itself or arisen outside the laryngeal skeleton, a firm or bone hard, small or large mass, which may be found to be contiguous with one of the laryngeal skeletal arches, may be palpable in the neck. Such a mass may not be tender and will move upon movement of the skeleton of the larynx. If the lesion involves or causes pressure upon the recurrent laryngeal nerve, as it did in the case presented in this paper, a vocal cord paralysis will be present. When the tumor is confined within the inner skeletal outline of the larynx, no palpable neck mass will be found. Inflammatory swelling can cause size variations of intraglottic lesions and also affect the size of those tumors confined outside the laryngeal skeleton.

If the glottis is examined by direct laryngoscopy, in those cases with an intralaryngeal lesion, a white or pale gray, firm or hard mass may be presenting itself in the lumen. Depending upon its particular location, a large tumor may cause a deformity and distortion of the outline of the glottic chink by encroaching upon the lumen of the larynx.

If the lesion has not broken through the mucous membrane lining the larynx, it will generally show no evidence of ulceration, and the markedly thinned overlying mucosa, which resulted from pressure atrophy, may reveal an exaggeration of its vascular supply, which may appear as several reddish vessels coursing over the mass. Ulceration may be present in those cases in which the overlying mucosa or tumor mass itself has been traumatized by previous biopsy attempts.

*Diagnosis.* Diagnostic difficulties often arise chiefly because of the infrequent occurrence of these tumors; therefore, they are sometimes not considered in the differential diagnosis. The diagnosis of these lesions is not often easy. Sometimes repeated biopsies by indirect and direct laryngoscopy techniques may be necessary, as negative biopsies may be obtained in those cases in which the overlying laryngeal mucosa is normal. Because of the firm hard nature of these tumors, a satisfactory biopsy specimen by the laryngoscopy technique may be quite difficult, and a thyrotomy may be required in order to adequately expose and biopsy them. A clinical suspicion of laryngeal chondroma may be considered if the history reveals a progressive hoarseness and slowly increasing dyspnea, and laryngoscopy reveals the presence of a smooth, hard, or firm intralaryngeal tumor without evidence of any ulceration. However, a diagnosis based on physical findings alone is only presumptive in most cases, and the definitive

diagnosis is made only by microscopic examination of the tumor tissue itself.

The diagnosis of laryngeal tumors has been aided considerably by roentgenography. The lateral view of the neck is quite helpful in determining the extent of the growth above or below the glottis.[9] There may be x-ray evidence of irregular calcium or bone deposits in the tumor suggesting the presence of a calcifying or ossifying chondroma (Fig. 1). Virchow[19] has stated that the process of ossification in a chondroma requires 20 to 30 years. Tomography is helpful in delineating laryngeal growths at different depths in the anteroposterior projection.[9] Foxwell[14] believes that it should be the rule to perform tomograms in any case of progressive dyspnea apparently unassociated with cardiac or pulmonary disease, as he was able to clearly show the presence of a tracheal mass (solitary tracheal chondroma) by this technique.

If the tumor has extruded itself or arisen outside the laryngeal skeleton, and has caused some compression of the esophagus, as a result of its progressive growth, a barium swallow may be helpful in revealing esophageal compression in the general area of the tumor.

*Differential Diagnosis.* Because of its infrequent occurrence, chondroma is seldom suspected as causing laryngeal symptoms. In their discussion of laryngeal chondroma, Ryan and Zizmor[9] stated that non-specific laryngitis, polyps, tuberculosis, . . . being more common, are usually suspected as the cause of hoarseness, dyspnea, and cough. The dyspnea . . . when complicated by upper respiratory infection, may simulate bronchial and cardiac asthma . . . or aspirated foreign body.

I think it might be appropriate here to make a few comments about chondrosarcoma (malignant analogue of chondroma) of the larynx as this condition, although extremely rare (only six cases of laryngeal chondrosarcoma reported in the literature), should be considered in the differential diagnosis of laryngeal cartilaginous neoplasms.

According to Willis,[1] there is sometimes no definite sharp line of separation between the solitary benign chondromas and the malignant cartilaginous tumors; growths of transitional type occur and occasionally tumors, originally thought to be benign, assume aggressive behavior.

Histologic changes of the cartilage cells making up these tumors may sometimes be the only differentiating factor between a benign

chondroma and a malignant chondrosarcoma. The cytology of the benign chondroma has been mentioned earlier in this paper; Lichtenstein[20] has remarked that a cartilage tumor is no longer to be regarded as benign if, when viable non-calcifying areas are examined, it shows, even in scattered fields: 1) many cells with plump nuclei; 2) more than an occasional cell with two such nuclei; and especially 3) giant cartilage cells with large single or multiple nuclei or with clumps of chromatin. In evaluating the cytology of cartilaginous tumors, attention should be concentrated upon areas which are viable and not heavily calcified or ossified. This is because the nuclei of the cartilage cells are likely to be swollen in those areas which are undergoing necrosis, heavy calcification, or ossification, and such changes are irrelevant to the benignity or malignity of the lesion.[20]

It has been reported that a chondrosarcoma not uncommonly results by a malignant transformation in the cells of a single benign chondroma or a cartilaginous exostosis.[20-22]

In contrast to chondromas, chondrosarcomas can metastasize, but this is usually a late phenomenon. Warren[23] reported a case of chondrosarcoma with intravascular growth and tumor emboli in the pulmonary circulation. Metastases occurring elsewhere than in the lungs are uncommon, but metastases to the liver,[24] bones,[25] and skin[26] have been reported. The extension of a chondrosarcoma to regional lymph nodes via lymphatics has occasionally been reported.

*Treatment.* The management of chondromas of the larynx depends upon their location and extent of growth. The consensus of opinion, among the investigators reporting these tumors, indicates that the only effective form of therapy and procedure of choice is surgery, as radium and roentgen therapy are of no value. (I might remark, however, that Huet[10] reported one case of laryngeal chondrosarcoma treated with radiotherapy with remarkably satisfactory results.)

A routine low tracheostomy should be done as a precautionary measure whenever a patient shows dyspnea and cyanosis due to the presence of a laryngeal chondroma. Link[8] believes that a routine tracheostomy should also be done in those cases in which there is a possibility of postoperative laryngeal edema, or whenever any portion of the cartilaginous framework of the larynx is sacrificed.

According to Gatewood,[27] direct laryngoscopy can be used to remove small chondromas confined to the arytenoid cartilage or epi-

glottis, but this technique is not useful for removing these tumors in other locations. A laryngofissure, with complete removal of the tumor, has been successful when the lesion is circumscribed and attached to the inner surface of the cricoid cartilage. Although it is rare, occasionally an encapsulated form of tumor, with a distinct line of separation between the growth and the original tissue, is found, and it can be completely "shelled" from its encapsulated bed. In such a case, it may not be necessary to remove the adjoining laryngeal cartilage.

As soon as the diagnosis is made, all cartilaginous tumors should be excised completely with a fairly wide margin in order to avoid recurrence,[5] and also because there is always a small percentage of these tumors which may assume malignant characteristics.[8]

According to Link,[8] tumors arising from the thyroid cartilage may be removed by an external approach with a submucous type resection or by morcellation without removing any of the laryngeal mucosa. The tumor bed should be well curetted if morcellation is the procedure selected.

A total laryngectomy should be done in those cases in which the tumor has obliterated the larynx, and it becomes impossible to carve a new larynx from the tumor mass. This procedure should also be done when the lesion has attained such a large dimension that its complete removal would sacrifice the laryngeal framework and result in collapse and stenosis of the larynx. When the cricoid ring is severed, stenosis is very apt to occur. However, Link[8] reported a case of laryngeal chondroma in which the entire cricoid cartilage was removed, half of the cartilage at an initial operation and the other half six years later with no resulting laryngeal stenosis. Rosedale[6] reported another case in which one-half of the cricoid cartilage and half of the first two tracheal rings were sacrificed, and then a prosthesis was used to prevent laryngeal stenosis.

Of all cases of chondroma of the larynx reported up to this time (including the case presented in this paper), there have been 24 laryngofissures and 16 total laryngectomies performed. A breakdown of these cases shows that in the series reviewed by Moore,[4] 39 operations were performed, of which nine were laryngofissures and six total laryngectomies. McCall, Dupertius, and Gardiner's[5] review showed ten laryngofissures and eight total laryngectomies. In Link's,[8] Rosedale's,[6] Moore's,[7] and Ryan and Zizmor's[9] cases, laryngofissures were done. In Huet's[10] case, the tumor was removed by way of a thyro-

crico-tracheotomy, and postoperative radiotherapy was given. Labayle's[10] case refused therapy. A laryngofissure was done in one of the three cases reported by Frenzel.[11] In Mattiolo's[12] case, a total laryngectomy was done. Dominik's[13] case refused operative treatment. A total laryngectomy was done in the case presented here.

After reviewing the literature, it appears that the total excision of laryngeal chondromatous lesions with an adequate wide margin is the only way to avoid a high recurrence rate. The cure rate after complete surgical removal of chondromas of the larynx should be good because these tumors are very slow growing and usually only locally invasive.

SUMMARY

Chondromas of the larynx are rarely encountered by the laryngologist, as evidenced by the small number of reported cases appearing in the literature. A review of the literature reveals 96 reported cases of chondroma of the larynx. Various pathological and clinical aspects of laryngeal chondromas are discussed, including the pathogenesis, gross and microscopic appearances, etiology, symptoms, physical findings, diagnosis, and treatment of these tumors. A review of the literature definitely indicates the tendency for these cartilaginous tumors to recur, sometimes in large bulk, if not completely removed; therefore, adequate surgery, with complete removal of the tumor, should be the initial procedure of choice if successful cure-rates are to be expected.

86 Piedmont Avenue

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LXXXI

# HEADACHES CAUSED BY BRUXISM

WOODROW S. MONICA, D.M.D.

ORANGE, N. J.

Bruxism, a term for abnormal grinding and clenching of teeth, causes prolonged and sustained contractions of the masticatory muscles. These forceful contractions exceed the physiologic function of the masticatory muscles and may result in partial tetany.[1] The pain that usually follows partial tetany may cause headaches.

The following two case reports suggest headaches caused by bruxism and a possible method for its control.

## REPORT OF CASES

CASE 1. A 30 year old married stenographer suffered every morning for three years from headaches in the frontal region. Her headaches started shortly after the insertion of two large gold dental bridges. She said her jaws felt tight in the morning. Several experienced physicians examined her and found her healthy except for occasional sinusitis. They offered her only temporary relief from the headaches.

Her mouth was clean, but the gums were inflamed from the excessive grinding pressure of bruxism. She had a severe malocclusion and a "chipmunk" look.[2] Roentgenograms of her teeth and temporomandibular joints showed no pathologic conditions.

I constructed an acrylic splint (similar to a boxer's mouthpiece) which she wore nightly for five weeks. During that time, she awoke free of headaches and with relaxed jaw muscles. Following instructions, she did not wear the splint for one week. Each morning thereafter, she awoke with headaches and a tense feeling in the jaws. Again she inserted the splint for five weeks and was asymptomatic. Upon further instructions, she did not use the splint for one week. During that interval, she awoke with headaches and tense jaw muscles. Then the patient wore the splint for eleven months and was symptomless except for several mild headaches during two sinusitis attacks. The inflamed gums and the "chipmunk" look disappeared.

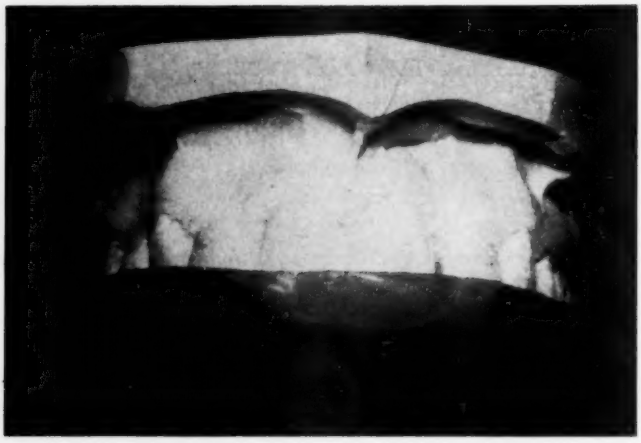

Fig. 1.—Acrylic splint.

CASE 2. A 45 year old married stenographer suffered from recurrent headaches in the frontal and left temporal regions for about ten years. Her headaches were more frequent in the morning and on week-ends. The headaches started after multiple extractions and the insertion of a partial upper denture. She had been treated at a headache clinic for three months, but had received only temporary relief.

The patient wore an ill-fitting upper denture. Her mouth was clean but her natural teeth showed excessive wear from bruxism. The masseter muscles were slightly pronounced. Roentgenograms of her teeth and temporomandibular joints were normal.

Like the previous patient, she also wore an acrylic splint for six weeks and was free of headaches. Following instructions, she did not use the splint for two weeks. During that interval, she suffered head pains and one severe headache. She wore the splint again for six weeks and was asymptomatic. Then she left it out for two weeks and suffered head pains and headaches. She continued to wear the splint for one year and was free of headaches except during several sinusitis attacks. The masseter muscles became less pronounced.

The following headache sufferer had a severe malocclusion, but he did not resort to bruxism. The splint did not relieve his headaches.

CASE 3. A 36 year old gardener suffered repeated headaches in the temporal and suboccipital regions for about twenty years. He was examined by many physicians and found physically well. They offered transient relief from his headaches.

He had a very clean mouth, healthy gingivae and normal tooth wear. The masseter muscles were not hypertrophied. Roentgenograms of his teeth and temporomandibular joints showed no pathologic conditions.

He wore an acrylic splint nightly and often during the day for one month, but he continued to have headaches.

## COMMENT

The physiology of the mandible suggests two basic considerations:

First, the mandible moves freely in all directions. Second, the mandibular teeth do not always contact the opposing teeth during mastication.[3]

When the free movement of the mandible is restricted by inadequate fillings, malocclusion or ill-fitting dentures, the subsconscious mind attempts to rub off or smooth the impediment. This forceful, non-functional grinding of enamel against enamel causes an abnormal stress to the masticatory muscles. When this activity persists, the overworked muscles may resolve into a partial tetanic contraction or trismus. Pain usually follows and may be referred to a homolateral area.

An acrylic splint is smooth and permits the easy movement of the mandible. Without the subconscious irritant of occlusal incongruities, the patient usually ceases to grind his teeth.

The excessively stimulated masseter muscles accumulate metabolites[4] that cause the hypertrophy or "chipmunk" look.

All patients with malocclusion do not grind their teeth nor do they suffer from headaches, per se. Likewise, all patients who habitually grind their teeth do not always have headaches.

310 MAIN ST.

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LXXXII

# STUDY OF SOME FEATURES OF THE NEURO-EPITHELIA OF THE INTERNAL EAR IN THE CHICKEN

Consuelo Savin V., M.S.

Mexico DF, Mexico

The study of the neuro-epithelia of the internal ear with the aid of the electronic microscope and under high power with the optic microscope has permitted us to observe important features of the structure of the ciliated cells and of the nerve endings reaching them.

Working on guinea-pigs, Jan Wersäll[1,2] saw in the cristae of the semicircular canals two types of ciliated cells (types I and II) related to two different types of nerve endings. Type I is composed of racket-shaped ciliated cells with a wide basal portion to which is connected a nerve ending divided into multiple branches forming a sort of enveloping chalice. Type II is formed by cylinder-shaped cells to which run nerve fibers terminated in a fine network with small buds at their ends.

Engström,[3,4] while studying the maculae of the saccule and the utricle and the sensorial epithelium of the cochlea in guinea-pigs observed both types of ciliated cells described by Wersäll. He also found two kinds of nerve endings in these cells: the granular and the non-granular which can only be seen under the electronic microscope.

The granular or slightly granular endings have in his opinion a probable afferent function, while the highly granular endings might represent the efferent nerve-endings with an inhibitory role.

Catherine Smith[5] confirmed these findings in the utricle of the same species.

The knowledge of the physiology and the pathology of the ear will progress with an increased knowledge of the histology and embryology of this region. The findings of the authors mentioned would certainly have greater value if found in other species, for which reason we have tried to find them in fowl.

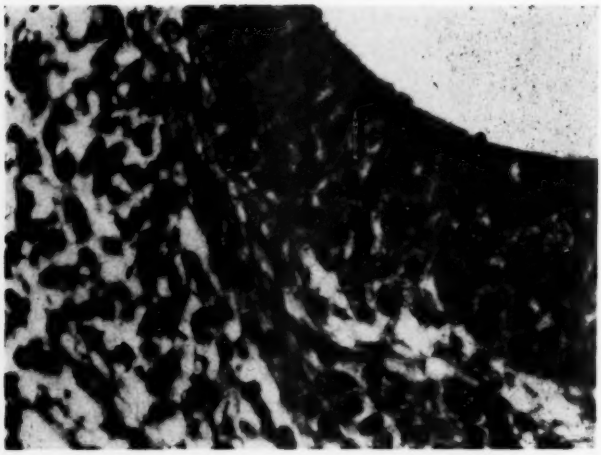

Fig. 1.—Fine argentophile filaments among the epithelial cells in otic vesicle in four-and-a-half-day chicken embryo.

## MATERIAL AND RESULTS

Embryos, fetuses and newborn chicks were used (Gallus domesticus). The smaller embryos had been incubated three days and newborn chicks were up to eight days of age. Embryos and fetuses were fixed in 10 per cent formalin Buin and acetic sublimate, the newborn chicks only in formalin. Fixation was carried out immediately after beheading the live specimens. After this, they were put into paraffin and the sections were stained with Harris hematoxylin and eosin and also with Palmgren's argentic impregnation.[6]

Observations were made with magnifications up to 3125 diameters in the optic microscope.

The study began with the examination of three-day old incubated embryos. In them the otic vesicle showed the beginning of a change in its general shape with a tendency to the initiation of the formation of the utricle and the saccule. The inner part of the vesicle showed an epithelial lining of greater thickness (the future crista) in the area adjacent to the neuroblasts which would form the ganglion. In the remainder the epithelium was thin. All this layer showed radially oriented cells arranged in three or four layers. Their nuclei were oval or round and caryokinetic figures were frequent.

In these three-day old embryos it was not possible to find nerve endings within the thickness of the epithelium despite the use of Palmgren's technique. Furthermore, the neuroblasts did not show peripheral dendritic prolongations.

At four and a half days fine argentophile filaments were discernible among the epithelial cells proceeding from the neuroblasts. As yet, no pericellular endings were seen (Fig. 1).

At six days of age the cristae are clearly seen within the endolymphatic spaces. The epithelium covering them was formed by prismatic cells quite similar to each other. The nerve fibers ascended to the highest part of the crista and an increase in their thickness became apparent.

At nine days the only difference was that nerve fibers were more numerous.

At eleven days the pericellular endings were seen for the first time. The nerve fibers divided themselvs into two branches in the vicinity of the ciliated cells and at their base they formed numerous ramifications which in some instances reached the upper third of the ciliated cell, surrounding it and ending in a point or in the shape of a bud.

The ciliated cells in the cristae tended to divide at this stage into two groups: Type A of large, clear round cells located mainly in the most row equator, of the same length as the previous but of lesser volume. They were found over the entire surface of the epithelium of the crista and they were more numerous.

At this stage there were many cells with intermediate characteristics which could be identified as belonging to either of the previous types.

All of the cells show under the optic microscope a single prolongation in the manner of an undulated cilium, wide at its base and two or three times the length of the cell (Fig. 2).

Between 14 and 18 days of incubation there appeared clear spaces surrounding some of the ciliated cells of both types; these spaces increased in number and size as age advanced.

Non-myelinic plexuses appeared in this stage at the base of the ciliated cells.

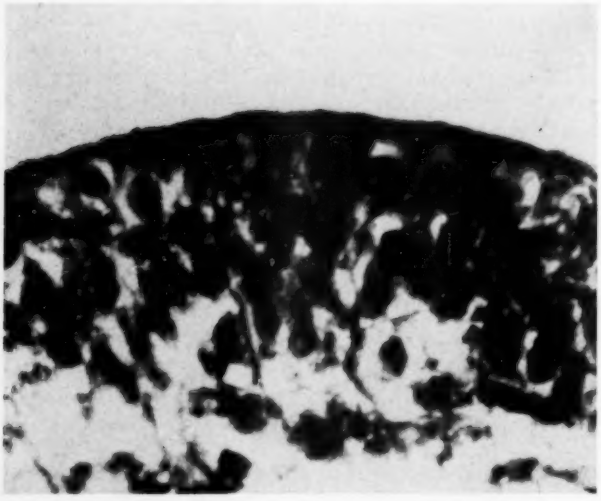

Fig. 2.—Ciliated cells in the cristae at eleven days chicken embryo. Observe the ciliated cells type "A" and "B".

In newborn animals the majority of the cells were well differentiated into types A and B, the former being the more numerous. The clear spaces which began appearing from the fourteenth day of incubation increased, containing up to five ciliated cells. They were provided with fibrillar formations, or at least bore this general appearance, and traversed the clear spaces in every direction. These clear areas were greater in number at the apex of the cristae.

In the areas of the cristae in fowl, known under the designation of lateral protuberances (Tetzius),[7] an undifferentiated epithelium was seen showing neither clear spaces, ciliated cells nor nerve endings.

Observing the lagenar epithelium we were able to see ciliated cells closely resembling each other without any clear spaces (Fig. 3). In the maculae, on the contrary, the clear spaces containing the sensitive elements were seen. Their nerve endings have the same characteristics mentioned for the cristae.

## COMMENT

In our material we found two types of ciliated cells as had been pointed out by Wersäll. However, the differences between types that

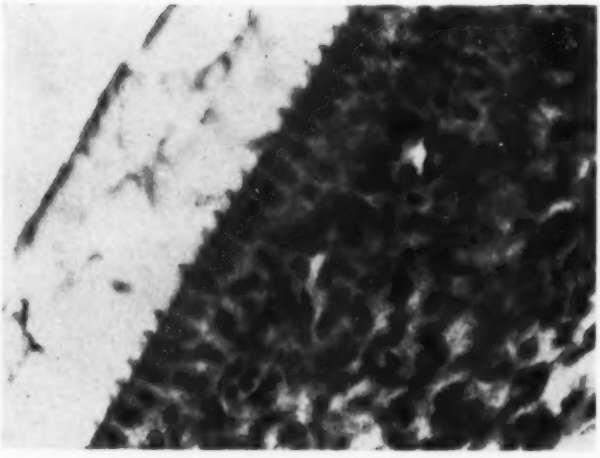

Fig. 3.—Lagenar epithelium with the ciliated cells closely resembling each other.

we call A and B are not as marked as those between type I and II in the guinea-pig. There are some elements with intermediary characteristics.

With the use of silver-stain techniques and under the optic microscope it may be ascertained that in the chick the nerve endings and the subcellular plexus are more numerous in type A than in type B.

The clear spaces surrounding the A type cells were found in the cristae and in the maculae as has been mentioned previously and not in the lagenar epithelium nor in the lateral protuberances of the cristae.

The interpretation of the significance of the clear spaces poses a delicate problem. Offhand one might think it is a technical artifact which took place perhaps during fixation and might have caused a retraction or vacuolization of the cytoplasm. Against this idea is the fact that these clear spaces appear progressively. They begin to be noticed in 16-day incubation animals and they increase in number and size up to the time of birth. Furthermore they are not found in the epithelial areas where the ciliated cells have not been differentiated into types A and B.

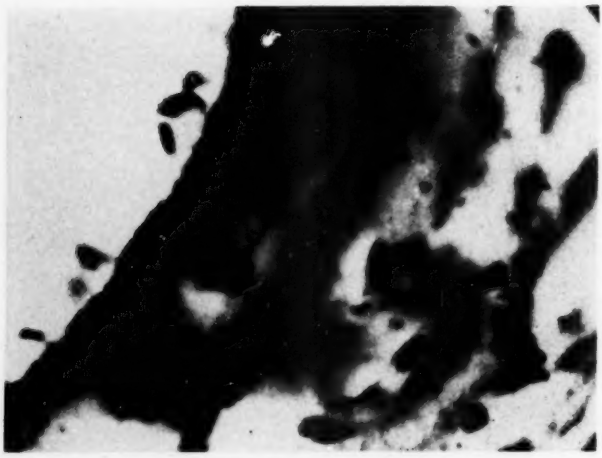

Fig. 4.—Nerve fibres traversing the clear space around the ciliated cell. Newborn chick.

Hilger[8] maintains that the epithelium of the fowl contains large amounts of water for which reason it does not keep well. This might be the explanation for this vacuolated aspect. Against this view it may be said that the nerve fibers traversing the clear space is proof to the contrary, since nerve endings are of necessity extracellular formations and not intracellular (Fig. 4).

We have made our observations in the ear of three newborn chicks, in two day old fetuses and in one 16 days old, which were studied separately. Perhaps with the use of other methods, especially of fixation and more particularly with perfusion, sufficient data may be obtained.

## SUMMARY

1. In chick-embryos, fetuses and newborn chicks two types of ciliated cells were found in the vestibular epithelia: one type was of large, rounded cells (type A), and the other of prismatic cells (type B).

2. The nerve endings of these two cellular types, as far as the optic microscope permits, are similar, showing slight differences. They

are more numerous and better defined in the round cells. At their tips are seen small buds, or else they end in a point.

3. The differentiation of the two types of cells appears in this species in nine-day incubation embryos and it is more marked later.

4. The nerve fibers reaching the sensitive cells of the neuroepithelium of static function appear in four-day-old embryos or at least this is the time when they become visible with Palmgren's method. At this age pericellular endings were not found. They appeared only in 11 day old animals.

5. The presence of clear pericellular spaces is described, especially surrounding the A type of ciliated cells. We ignore their significance. These clear spaces are not present in the epithelium of the lateral protuberance of the cristae nor of the lagena.

6. A and B cells were not seen in the lagena.

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The author wishes to express her gratitude to Dr. A. Palmgren for his valuable aid in the preparation of the silver stain technique used in this work.

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LXXXIII

# DEAFNESS FOLLOWING SHORT-TERM EXPOSURE TO INDUSTRIAL NOISE

S. L. Shapiro, M.D.

Chicago, Ill.

The medical literature on acoustic trauma can be divided into two parts: those papers which deal with explosion or blast deafness, and those which concern themselves with the effect of chronic exposure to industrial noise upon the ear.

The salient features of the first variety involve a sudden injury due to the conversion of a solid or liquid into a gas with the creation of an enormous pressure wave or shock pulse preceding the sound of the explosion, both factors combining to produce the damage. The majority of the articles on this type of deafness came during or after World War II and deal with the effects of war explosives. The classic paper on this topic was presented by Dr. Henry Perlman and is a remarkably clear exposition of the mechanics, physiology and pathology of explosion injury to the ear.[1]

The second type of acoustic trauma, that resulting from chronic exposure to industrial noise, had been of interest to otologists for many years. A considerable European literature on the subject had accumulated by the first World War and since that time many excellent articles have been published in this country and abroad dealing with the effects, causes and prevention of hearing impairment in noisy occupations. Particularly worthy of mention are the concerted efforts represented by a number of recent annual symposia on Noise as well as by the work of a subcommittee of the American Acoustical Society and, of course, by the subcommittee on Noise in Industry of the Academy. In this field as well, important contributions have been made by Dr. Meyer Fox of Milwaukee.

My purpose is to call attention to a third form of acoustic trauma which has received very little mention in the literature. This condition which I shall call "Deafness Following Short-term Exposure to

Read before the Chicago Laryngological and Otological Society, April 7, 1958.

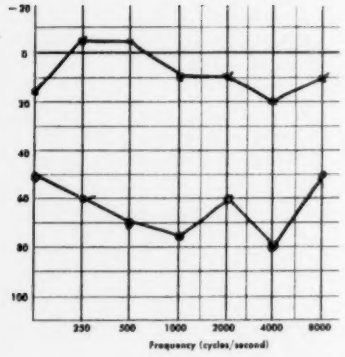
Industrial Noise" is uncommon and occurs only under quite special circumstances but its end-result, in my experience, has been a true clinical deafness for the ear involved in terms of ability to carry on an ordinary conversation as well as in the accepted standard of pure tone impairment.

My material consists of six patients, all of whom were seen for insurance companies representing the employer. Routine investigation of the facts relating to the injury, including as far as possible the previous state of the hearing, was made in each instance by the company and at least one other otologist besides myself examined the patient. My own examination of the earlier patients was made with the D-5 Model Maico audiometer, of the more recent ones with the new MA-1 Maico. The "normal" threshold level for each instrument was calibrated by comparing it with that of a number of young adults and it was found to approximate the zero line on the audiogram. The tuning forks used were those of the large Riverbank set supplemented by the large Bezold-Edelman A fork for the Schwabach and Weber tests; a traditional technique with duration of the responses timed in seconds was used for the fork tests. Speech tests were confined to the residual whispered voice test. A malingering test was done on each patient.

## REPORT OF CASES

CASE 1. L.P., a 36 year old construction worker, was seen on December 22, 1952. He stated that two days previously, after working for an hour and a half in a deep caisson hole with a pneumatic air spade, his right ear blocked up and he is now unable to hear with it. The nose and throat examination showed an acute rhinopharyngitis; the right tympanic membrane showed some scarring as well as some bulging inferiorly, the left one was normal. The patient denied any previous hearing difficulty with either ear but admitted having had a cold for about a week. The right eustachian tube was inflated and he was told to return in four days. At the second visit the appearance of the nose and throat was much improved and the bulging of the right drum was gone; the eustachian tube was patent. Hearing tests at this visit gave the results shown in Figure 1.

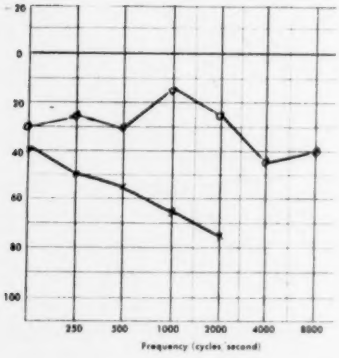
The man's hearing failed to improve during the succeeding months and six months later he was examined by another otologist whose audiometric and voice tests indicated no change from the findings just demonstrated and confirmed his claim of deafness of the right ear. There is admittedly some uncertainty with respect to the role




| R. | | L. |
|---|---|---|
| 0′ | Whisp. | 20′ |
| | ↑ Weber | |
| 35″/21″ | Rinne | 95″/53″ |
| | Schwab. 27″ | |
| D72 | Low T. | G24 |
| 14″ | High T. | 25″ |

Conv. maling. tests negative
Recheck six months no change.

Fig. 1.—L.P., age 36. Deafness right ear after exposure for an hour and a half to air hammer noise.



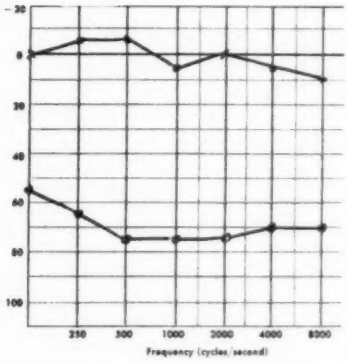

| R. | | L. |
|---|---|---|
| 9′ | Whisp. | 0′ |
| | ←Weber | |
| 68″/30″ | Rinne | 18″/10″ |
| | Schwab. 26″ | |
| C32 | Low T. | C64 |
| 18″ | High T. | 7″ |

Conv. maling. test negative.

Fig. 2.—N.F., age 30. Deafness left ear after exposure for eight and a half days to air hammer noise.

of the "cold" in this instance but I have included this case with the others because of the marked perceptive factor indicated by the functional tests as well as in view of my previous and subsequent experience with this particular type of acoustic trauma.

CASE 2. N.F., a 30 year old construction worker, was seen on November 14, 1953. He stated that six months previously he had




| R. | | L. |
|---|---|---|
| 0′ | Whisp. | 18′ |
| | ↑ | |
| | Weber | |
| 0″/18″ | Rinne | 91″/51″ |
| | Schwab. 30″ | |
| 0 | Low T. | G24 |
| 0 | High T. | 26″ |

Re-check month later same.
Stenger test neg. maling.

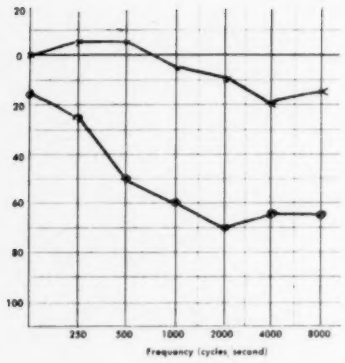
Fig. 3.—M.H., 42 years old. Deafness right ear after exposure to air hammer noise for two days.

worked for eight and a half days with a pneumatic hammer in a narrow space, holding the tool on his left shoulder; at the end of this period he noticed that the hearing in his left ear was bad and now it is gone entirely. Previous history as to the ears was negative. Examination showed a normal nose and throat; the tympanic membranes were retracted with absent light reflex but otherwise negative. Functional tests were as shown in Figure 2.

This patient was seen by another otologist who confirmed the finding of moderate hearing impairment on the right side and clinical deafness on the left.

CASE 3. M.H., a 42 year old construction laborer, was seen on March 11, 1954. He stated that about one and a half months ago he ran an air hammer for two days working in a round hole about 20 feet wide and 20 feet deep. At the end of this period he found that he was deaf in the right ear. He has since seen a number of doctors and also has gone to Hines Hospital, as he is a veteran, but he still cannot hear with this ear. Examination of the nose, throat and tympanic membranes gave normal findings. Hearing tests were as shown in Figure 3.

A re-check on March 26, 1955, a year later, showed that the man was still deaf in the right ear. A Stenger test for malingering was negative. This man was examined in 1954 by the audiologic service




| R. | | L. |
|---|---|---|
| 1′ | Whisp. | 12′ |
| | ↑ | |
| | Weber | |
| 51″ | | 89″ |
| 35″ | Rinne | 38″ |
| | Schwab. | |
| | 25″ | |
| G48 | Low T. | C32 |
| 10″ | High T. | 27″ |

Re-checks one and four months later showed no change. Voice maling. neg.

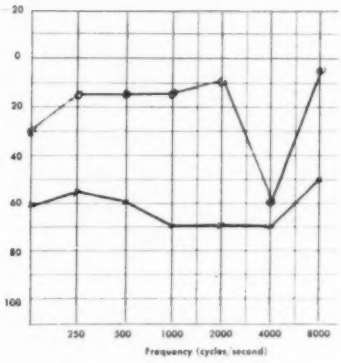
Fig. 4.—A.B., 33 years old. Deafness right ear after exposure to continuous sledge hammer noise for one day.

of one of our large hospitals who confirmed the extent of the impairment for the right ear.

CASE 4. A.B., 33 years old, was seen on February 17, 1954. He stated that he was normally a machine operator but eight days previously had substituted for another worker on a different job, beginning at 8:00 A.M. and working until 4:00 P.M. His task for that day consisted of pounding certain types of a fitting with a sledge hammer, working on his knees with his head about two feet away from the fitting which he was striking. At end of the day he noticed that he had difficulty in hearing, mostly on the right side. The left ear has since cleared up but the right ear is still quite deaf. Examination indicated the nose and throat to be negative; the tympanic membranes were retracted but otherwise negative. Hearing tests were as shown in Figure 4.

Re-examination one month and again four months later showed no essential change. Examination by another otologist showed approximately similar findings.

CASE 5. J.J., a 43 year old construction worker, was seen on October 15, 1955. He stated that a month previously he ran a pneumatic hammer for three or four days cutting concrete, after which he noticed trouble in hearing and noise in left ear which has persisted to date. He denied any previous trouble with his ears. Examination




| R. | | L. |
|---|---|---|
| 9′ | Whisp. | 0′ |
| | ↑ | |
| | Weber | |
| 80″/31″ | Rinne | 22″/20″ |
| | Schwab. 18″ | |
| C32 | Low T. | A53 |
| 18″ | High T. | 7″ |

Re-check 1-16-56 another otologist no change.
Conv. maling. test neg.

Fig. 5.—J.J., 43 years old. Deafness left ear after exposure to air hammer noise for three or four days.

showed the nose, throat and tympanic membranes to be entirely normal. Hearing tests were as shown in Figure 5.

This man was not seen by me again but was examined three months later by another otologist whose audiometric and voice tests gave results similar to those indicated above.

CASE 6. W.F., 33 years old, was seen on May 4, 1957. He stated that he was a grinder. While working inside a tank four weeks previously, someone struck two heavy blows with a sledge hammer on the side of the tank next to his left ear. He immediately became dizzy and nauseated, left the tank and was sent to a doctor and later to an otologist. Since the occurrence he has had a hissing sound in the left ear and poor hearing on this side. Examination showed the nose and throat to be negative; the tympanic membranes were considerably retracted but otherwise negative. Hearing tests were as shown in Figure 6.

A test with the Frenzel glasses showed a mild horizontal nystagmus to left in the supine position.

The man was not seen again by me but was examined five months later by another otologist whose tests showed no improvement. This appears to be an instance of a strong acoustic insult which was too sudden for the intratympanic muscles to exert their customary pro-

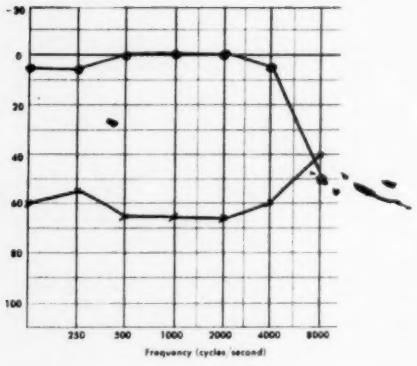
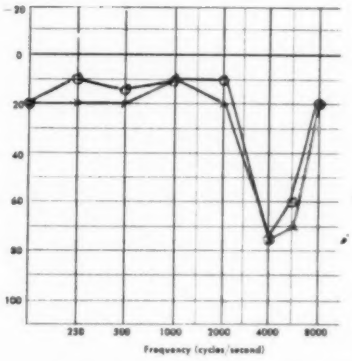



Fig. 6.—W.F., age 33. Deafness left ear after exposure to noise from sledge hammer blows on the outside surface of a metal tank next to his left ear.

tective function. I might add that this is the only case of the ones being reported for which I found any parallel in the literature.[2]

In going over the preceding histories it can be readily seen that they possess certain features in common. None of the men were older than 43, four of them were in their thirties. None were steady workers with noisy equipment; as a rule the particular job involved was performed in a small space with the noise close to one ear. As to the source of the noise, two involved sledge hammers, the rest came from pneumatic tools. The noise from the latter comes from both the tool and the object which it strikes, reaches a level as high as 125 db and is a broad band type of noise, ranging from 300 to 4000 cps with most of the frequencies in the highest or most objectionable portion of the spectrum. I might add at this point, that another case which I examined about 20 years ago, also resulted from the use of a pneumatic hammer. My record for this man has been lost but I recall that it involved a young construction laborer who worked with this tool in a caisson foundation for several days and wound up with one deaf ear.

As indicated previously, the results of the hearing tests are in all instances those of a high-grade perception deafness. There is nothing in the audiogram of any impairment due to noise to distinguish it from an inner ear involvement due to other causes but there are some




Chronic noise exposure.
Gear cutter; machine shops
15 years.
No hearing complaint;
some reduction forks and
whisp. voice.

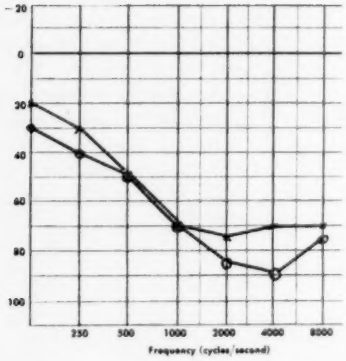
Fig. 7.—R.B., age 37. Bilateral high tone loss due to fifteen years of exposure to noise as gear cutter.

differences between the condition which is under discussion here and that due to chronic noise exposure. A glance at three audiograms illustrating the latter will serve to make the point.

CASE 7. (Fig. 7). This is the audiogram of a 37 year old man who has been working as a gear-cutter for 15 years; it represents one type of change following chronic exposure to noise. In contradistinction to the cases which have been presented previously, the impairment is bilateral, symmetrical and partial. These characteristics are true of the vast majority of hearing losses due to chronic noise exposure.

CASE 8. (Fig. 8). This is the audiogram of a 58 year old man who worked with drop forge hammers for 15 years; in 1952 he had a mild coronary attack and changed to a lighter job in the same tractor plant. He stopped working in 1955 when he found that he could not hear and has worn a hearing aid since that time but complains that he still hears badly. This is one of the few cases of chronic noise exposure I have seen in which the impairment reached the state of deafness; none of these men has been under the age of fifty.

CASE 9. (Fig. 9). This is the audiogram of another man who also happens to be 58 years old. He worked with drop forge hammers for 28 years including several years in the same plant as the previous patient. He had no complaint whatever about his hearing, being seen for another condition. If one makes proper allowance for presbyacusis in this case there is hardly any impairment at all due to noise.




Chronic noise exposure.
Drop-forge hammers 15 years.
Using hearing aid but of little value.
Whisp. voice six inches, tuning forks in accord.

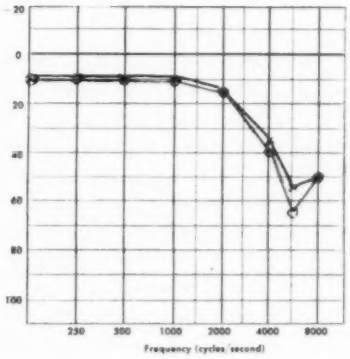
Fig. 8.—B.F., age 58. Bilateral deafness. Worked with drop hammers for fifteen years.

Neither of these two patients used ear protection while at work, and both of them have been away from their occupation for a considerable period. The striking difference in the state of their hearing cannot be accounted for except on the basis of a marked difference in susceptibility to injury by noise.

## COMMENT

It is my belief that this factor must also be considered in discussing the cases of short-term exposure to noise which form the subject of this paper. When one has taken into account the unfavorable circumstances which have been mentioned, there still remains the fact that operations with hammers, especially of the pneumatic type are fairly common in modern industry even under the conditions stated in my histories. It seems a fair assumption that an unusual susceptibility to noise trauma must have existed in at least some of these men.

This aspect of the problem is still largely shrouded in mystery. We are unable to state definitely which portion of the acoustic mechanism is involved and, in spite of a good deal of experimental and clinical research, have as yet no reliable method for predicting in advance any special liability on the part of an individual to permanent damage by excessive sound. A number of years ago Peyser,[3] a German otologist with considerable industrial experience, suggested using induced auditory fatigue as indicated by the extent of the temporary




Chronic noise exposure.
Drop-forge hammers 28 years,
no protection.
No complaint about hearing.
Slight reduction tuning forks
and whisp. voice.

Fig. 9.—O.U., age 58. Exposure to noise from drop-forge hammers for 28 years.

threshold shift following a measured acoustic stimulus, as a means of screening people who would be likely to receive abnormal damage to their hearing from occupational noise; research along this line was continued with various modifications by a number of men here and abroad. One of the latest and best articles on the subject by a group of French otologists[4] involved careful experiments with auditory fatigue tests both in normal persons and in groups of men who had served for varying periods in noisy industries. These experiments showed indeed, as found by others, that from the standpoint of auditory fatigue following experimental stimuli there was an average group, a hypersusceptible group and a small insusceptible group; when these results, however, were singled out in the group of people who were already working in a noisy occupation, it was found that no correlation could be established between the experimental findings and the actual effects of the chronic exposure as indicated by the audiograms of the workers. In a very recent study of the existing tests for susceptibility, Summerfield, Glorig and Wheeler[5] reached a similar conclusion.

There is also the interesting question of adaptation to noise. On purely clinical grounds there appears to be a strong possibility that, just as there are people who possess a natural resistance to acoustic injury as exemplified by the audiogram of the second drop-forge worker shown previously, a certain unknown percentage of persons can acquire some resistance to noise injury over a period of time, pro-

vided that the initial sound is not too powerful. Presumably this would involve better functioning of the intratympanic musculature.

The question arises as to what measures can be used to prevent such aural disasters as the ones which have been described. Pre-employment audiograms followed by a re-examination at stated intervals are of value in detecting unusual susceptibility among workers in chronically noisy occupations. The men who form the subject of my report do not, however, fall into this category of employees. They might be described as random workers with noisy tools under unusually hazardous conditions. The day has hardly arrived when construction workers and casual laborers will receive routine pre-employment audiograms, but even if this were done, given the conditions of their work, follow-up tests could hardly be made in time to do any good. There are, however, two things which could serve to minimize the danger of deafness when pneumatic or other noisy tools are used. The first is the reduction of the noise at the source, especially of the pneumatic tool which is the chief offender. This is actually being done by one manufacturer of these instruments and the process is described in a scholarly article by the chief engineer of the company in a recent issue of Noise Control.[6] The most practical measure, however, would be to require construction and other companies to furnish the best ear plugs or other protective devices to workers engaged in these hazardous tasks.

A final word is in order. The subject of noise in industry is a live one in this country with important medical, social and legal aspects. A great deal of valuable work has been done and is in process of accomplishment by members of the subcommittees previously mentioned as well as by others in order to shed light on the questions and possible solutions in this field. This work is in part experimental, in part clinical, in large part statistical dealing with problems of safe intensity and frequency levels for different types of industrial noise, the effect of duration of exposure in different occupations, the relationship of presbyacusis and other phases of the noise problem. I do not feel that the case reports which I have presented negate in any way the results of those studies on the effect of chronic exposure to noise which deal with large groups of workers and strive to present an average or composite picture. It seems to me that these histories serve rather to stress facts already known in relation to damage from chronic exposure since they present situations in which by virtue of the narrow confines of the working space and closeness of the source of the noise, its intensity at the ear of the worker must have been greatly magnified; above all this contribution may serve as another reminder

of the importance of that unusual liability to damage by noise which resides in certain people and which can modify considerably our accepted standards for this type of injury.

25 E. Washington St.

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LXXXIV

# BILATERAL TEMPOROMANDIBULAR INTRA-ARTICULAR ANKYLOSIS FOLLOWING RHEUMATIC FEVER

REPORT OF A CASE

ALVIN C. POWELEIT, M.D.
COVINGTON, KY.
AND
ROBERT C. KRATZ, M.D.
NEWPORT, KY.

In the course of treatment of temporomandibular ankylosis, certain interesting corrective measures have been noted which we should like to present. At the outset let us clarify some of the present terminology relating to this condition.

1. Intra-articular, or true bony ankylosis: the osseous union lies within the joint itself.

2. Extra-articular ankylosis: the ankylosing factor is due to induration of the surrounding tissues.

Both types are defined in degree as complete or partial, and the involvement may be unilateral or bilateral. The underlying causes of ankylosis are trauma or disease. Surgery[1] is the only available effective treatment in true bony ankylosis; stretching or forcefully dilating the jaws will aggravate this condition.

Various methods[2] employed in the treatment of intra-articular ankylosis have included arthrolysis (division of the joint surfaces); arthroplasty (reconstruction of a new joint at the level of the neck of the condyle); and osteo-arthroplasty (excision of the head and neck of the condyle). Our approach to the problem was based on

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From the Department of Otolaryngology, Booth Memorial Hospital, Covington, Kentucky, and the Department of Otolaryngology, the University of Cincinnati College of Medicine.

three predominant factors: 1) osteo-arthroplasty with excision of the head of the condyle; 2) an attempt to preserve the joint capsule; 3) elimination of the use of a fascial graft between the cut end of the condyle and the synovial cavity.

The following is the report of a case wherein such a procedure was done on a patient who had had a ten year history of temporomandibular ankylosis following rheumatic fever. Mobility became increasingly limited between the ages of 12 and 14, with incipient temporomandibular rigidity. The patient finally came under our care when he could no longer open his mouth wide enough to permit the passage of an aspirin tablet. He was admitted to the hospital on February 2, 1958.

Physical Examination: Revealed a normal-appearing male, not acutely or chronically ill, who could open his mouth only 7 millimeters. X-ray examination of the temporomandibular joints suggested osteomatous growths about the head of each mandible.

Impression: Bilateral temporomandibular ankylosis following rheumatic fever.

A two stage procedure was decided upon in an attempt to free the ankylosed structures.

I. With a tracheotomy set available, the patient was given a small amount of pentothal anesthesia and a screw type mouth gag was placed between the molars. The jaws were then forced open 2 centimeters.

II. Two days following the first procedure, the pharynx was anesthetized topically with 10 per cent cocaine. Using a DeVilbiss spray, an endotracheal tube was passed through the nose into the larynx and general anesthesia was begun. The line of incision was started directly in front of the ear, just anterior to the tragus and extended downward as far as the lobe. The joint was approached through the plane between the parotid gland and the external auditory canal, thus avoiding the facial nerve. The zygomatic process of the temporal bone was exposed and the joint and head of the mandibular condyle were located just below and behind the zygomatic arch. An incision was then made through the periosteum of the zygomatic area and the region of the condyle was exposed subperiosteally.

With up-cutting Kerrison punch forceps and a chisel, the head of the condyle was removed. The major portion of the articular

capsule was preserved, thus avoiding possible damage to the internal maxillary artery located just medial to the neck of the condyle. No fascia or muscle were placed into the defect. The wound was then closed using several 00 plain gut sutures to close the deeper structures and fine silk in the skin. A pressure bandage was applied for 48 hours.

Four weeks later the same procedure was carried out on the opposite side. (No attempt should be made to operate on both sides simultaneously because the masseter muscles pull the mandible upward, producing malposition.)

The patient is now able to chew without difficulty.

SUMMARY

A case is reported of a patient who had had a ten year history of intra-articular temporomandibular ankylosis following rheumatic fever. The preferred method of treatment was osteo-arthroplasty with excision of the head of the condyle, preservation of the major portion of the joint capsule, and avoidance of the use of a fascial graft between the cut end of the condyle and the synovial cavity.

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LXXXV

# TWO NEW METHODS FOR THE ASSESSMENT OF CENTRAL AUDITORY FUNCTIONS IN CASES OF BRAIN DISEASE

JOSEPH MATZKER, M.D.

MAINZ, GERMANY

In contrast to the assessment of peripheral auditory impairments, methods for evaluation of central hearing deficiencies are not well developed. In 1957, Goodman[5] called attention to the fact that tests specifically designed for the assessment of central auditory impairment are largely lacking. Considering the auditory process as a whole one must admit that the central portion of the auditory system plays a decisive role. We believe therefore that the assessment of pathological changes within the central auditory pathways will gain more and more importance in the future. To this end, it became mandatory to develop new methods of auditory testing. Present-day hearing tests (threshold audiograms, tuning fork tests, speech tests, recruitment measures) are not designed to give information about the state of the central portion of the auditory system. The majority of central auditory lesions in our experience cannot be assessed to any degree of certainty with the aid of standard audiometric methods. Patients with such central lesions are often judged by the otologist as having essentially normal hearing. However, there are cases of brain tumors which show certain aberrations in hearing such as difficulties in following verbal communication in noisy environments. It stands to reason that these could perhaps be uncovered by specially designed audiometric tests.

Within recent years, Bocca[1-4] (Milan) developed and tested some new methods for the assessment of central auditory functions. These methods, which are well known in the U. S., include the so-called accelerated, interrupted, and artificially scanning speech, the alternating speech, and the "binaural summation and integration." Bocca's methods have made it possible to pinpoint many more central auditory

From the Department of Oto-Rhino-Laryngology, Johannes Gutenberg University, Mainz (Rhine), Germany.

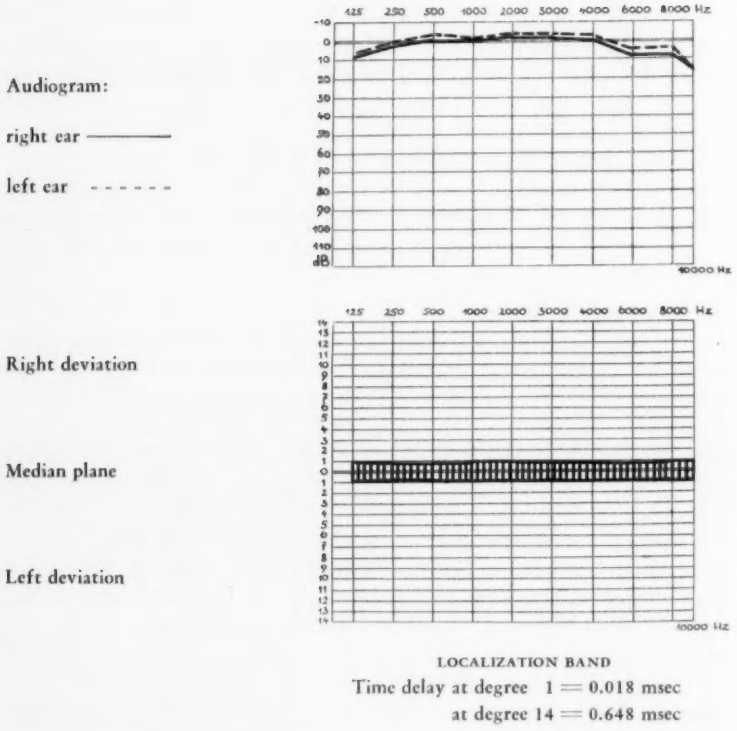


Fig. 1.—Average values of 12 normal persons between 21 and 30 years. The shaded zone corresponds to the middle impression and forms the localization band. Here, the band is narrow and lies on the zero line.

lesions than were heretofore feasible, especially in cases of brain tumors.

Independently from Bocca's work, Sanchez-Longo and Forster[12] have made a study of the ability to localize sound in patients with brain tumors. Using "white noise" as their test signal, these authors demonstrated that in patients with tumors in the temporal lobe a sound source is lateralized to the contralateral side. However, in patients with lesions in other cerebral regions no aberration was found in their ability to localize sound. Other than the report by Sanchez-Longo and Forster, who are neurologists, we were unable to find any reference to attempts by otologists of using the ability to localize

sound for the purpose of uncovering central lesions. It is our intention to show in the present paper that a) the ability of binaural auditory fusion and b) that of sound localization are excellent indicators of specific pathological changes in cases of several different brain lesions, since both of these are central auditory functions.

## OWN METHODS OF CENTRAL AUDITORY TESTS

a) *The "Binaural Test."* The test is based on the principle of binaural fusion. Over a period of three years and after examining more than one thousand persons, we were able to prove to our own satisfaction that the binaural test explores specifically the integration, which takes place in the brain stem, of signals traveling in the bilateral auditory pathways. This work was carried out in the Dept. of Otolaryngology of the University of Mainz. The following is a general description of the test method. Two narrow band-pass filters, a low one from 500 cps to 800 cps and a high one from 1815 cps to 2500 cps are used and speech signals are transmitted through both of these filters. Each band by itself is too narrow to allow recognition of the test words. However, when both bands are presented together, adequate recognition is possible. The bands are now separated in such a way that the right ear receives only the low-frequency band, while the left ear receives the high-frequency band. Each ear is presented with only one phonetic fragment of each test word, and yet there is excellent integration of the two fractions indicated by a near optimal score when the test is given to normal hearing persons. This integration is believed to take place within the brain stem where the two auditory pathways from either side are interconnected. Normal recognition of the test words then appears to indicate good functioning of the synaptic connections in this region. If, however, the discrimination score is below normal (or the patient fails altogether in this test) the synaptic function within the brain stem is judged defective. This failure of bilateral integration is probably caused by the loss of ganglionic cells within the primary auditory centers, specifically at the level of the cochlear nuclei and of the medial geniculate bodies.

The actual testing is arranged as follows. The patient is first acquainted with the requirements of binaural hearing. This is achieved by letting him answer ten carefully phrased questions transmitted to him through a set of ear phones. Next, the patient listens to a series of 41 two-syllabic words (phonetically balanced) which are transmitted in binaural fashion as described above. Mistakes are judged only from the phonetic standpoint whereby importance is placed upon the correct recognition of the vowels. This first test is

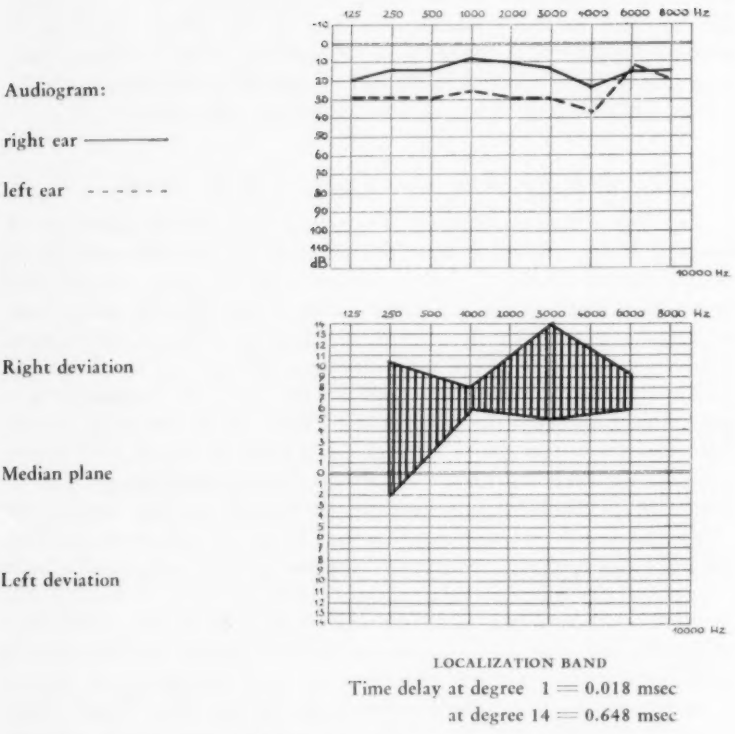


Fig. 2.—Localization band in a woman with a great frontal meningioma on the left side.

followed by a second one, again a series of 41 words which are phonetically balanced. This series is given so that both frequency bands are received in either ear, that is, in a diotic manner of presentation. Each ear receives now both phonetic fractions together so that the brain stem is relieved of its task of binaural fusion. As a rule, the words are now well recognized.

Upon completion of the second test, a third list of phonetically balanced words is presented, again in the same binaural fashion as in the first test. This is done in order to force once more bilateral integration at the level of the brain stem. A patient with normal brain function makes now even noticeably fewer mistakes than in the first binaural test run. In contrast, patients with brain lesions make

a high score of mistakes, definitely higher than in the second (diotic) run because of their failure of bilateral integration.

Normal test results, expressed as the number of mistakes in the three test runs (binaural-diotic-binaural) may look as follows: 9 - 4 - 3; 14 - 6 - 6; or 12 - 3 - 4. In contrast the following results are indicative of pathological changes: 27 - 4 - 12; 31 - 7 - 18; 25 - 4 - 13; or 37 - 24 - 38. It goes without saying that each examiner must gain experience until he is able to tell mistakes and to judge the test results properly.

Our examination of more than one thousand patients and test subjects showed pathological test results in:

| | |
|---|---|
| Cerebral tumors, all brain regions | 84% |
| Cerebral atrophy of different causes | 69% |
| Hypertension, all age classes | 46% |
| Multiple sclerosis | 35% |
| State after skull trauma | 30% |
| Endogen psychosis | 0% |

(NOTE. The percentages given represent those of the number of cases tested in each category.)

We are able to support the assumption expressed previously of a failure of bilateral integration at the level of the brain stem by histopathological findings. Some of our patients who had shown abnormal results when performing the binaural test died from their underlying cerebral lesions. After autopsies had been obtained, Dozent Dr. J. Ruckes[11] (Institute of Pathology, Mainz University) was able to demonstrate histologically changes in the brain stem of such patients, particularly within the olivary region. These changes included microhemorrhages, with capillary thrombosis, edema, and degeneration of ganglionic cells in all stages up to a complete necrosis. Dr. Ruckes explains these changes by vascular reflexes in the sense of Ricker.[10] The heaviest concentration of such vascular changes may be in the regions remote from the auditory pathways, for instance, in the case of tumors within the frontal or the occipital lobes. Dr. Ruckes was able to confirm his theory with the aid of animal experimentation as

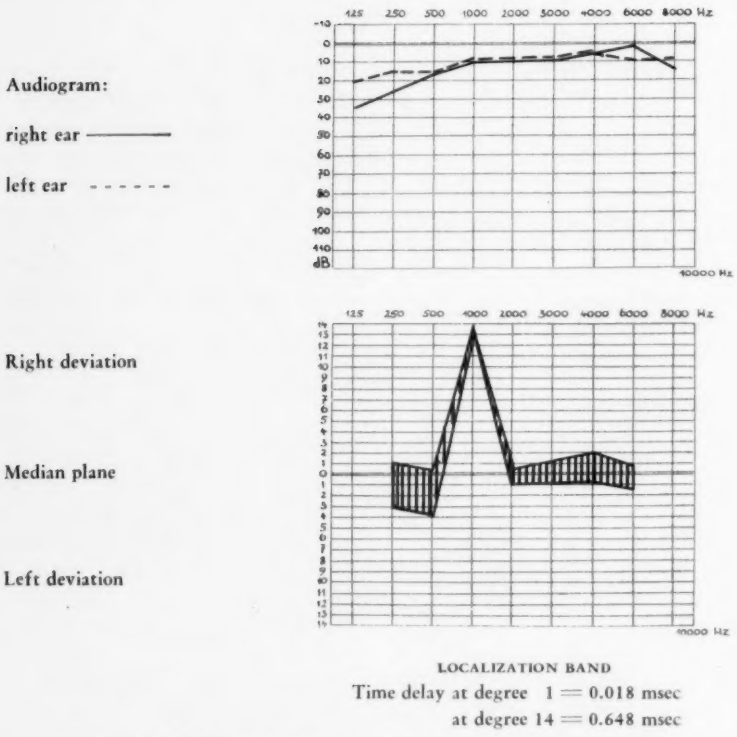


Fig. 3.—Localization band in a girl with a great temporal meningioma on the left side. The sound impression is only deviated in one frequency (1000 cycles); in all other frequencies sound localization is normal.

well as by examining several hundred human brains. The assumption is justified that the vascular disturbances (in the sense of Ricker) cause a functional failure of the ganglionic cells in their region. If thus the synapses in the brain stem are blocked, the integration of signals traveling in the two bilateral auditory pathways becomes impaired. It appears therefore that the binaural test is a reliable indicator of the extent to which ganglionic cells within the brain stem are out of operation. This concept makes it plausible that the physiological loss of ganglionic cells occurring with age should cause some failure in the binaural fusion between auditory signals. This is borne out by the finding that in persons over 65 years of age the binaural test tends to become positive, nearly without exception. We wish to make one point in this connection. The hearing impairment due to age,

presbycusis, is in our opinion more dominated by the central than by the (recognized) peripheral changes.*

b) *A Test of Sound Localization Using a Method of Interaural Time Differences.* For a period of more than a year, we have studied the ability to localize sound in patients with intracranial lesions. However, in contrast to Sanchez-Longo and Forster, we did not employ a free-field testing technique, which in our opinion has potential sources of error. Instead, we utilized interaural time differences which were electrically generated. Furthermore, we did not employ white noise as test signals as Sanchez-Longo and Forster did in their study but pure-tone signals of frequencies between 125 cps and 8000 cps which were interrupted at regular intervals. This way we feel we obtained more detailed information.

In order to achieve interaural time delays of known duration, a delay line of 36 sections was used. The signal itself was generated by a Peters audiometer, type SPD 2 (manufactured by Alfred Peters, Sheffield, England). The smallest time interval the delay line was capable of producing was 0.018 msec. This interval is already large enough to cause, in normal-hearing persons, a definite lateralization of the imaginary sound source to the side where the signal arrives first. The greater the time difference, the farther lateral of course the sound seems to move. At an interval of 0.648 msec, eventually, the impression obtained that the sound source had moved to a position of exactly 90° off the median plane. This final figure approaches the Hornbostel-Wertheimer constant, the value of which is ordinarily given as 0.6363 msec. The difference between that and 0.648 msec, the value obtained without experimental set-up, is negligible.

The tone is interrupted eight times per second as facilitated by the Peters audiometer. Unfortunately, the switching by this instrument is not entirely free of audible transients. The potential significance of this point will be discussed later. Approximately three or four "on-periods" constituted one test run. This limitation was found necessary in order to prevent auditory adaptation or fatigue. Therefore, test runs were separated from each other by brief rest periods of 15 seconds duration each.

The level of presentation varied somewhat with frequency for technical reasons. In the range from 500 cps to 2,000 cps it was

*For further information concerning the binaural test, reference is made to the monograph: "Ein binauraler Hoersynthese-Test zum Nachweis zerebraler Hoerstoerungen," Geo. Thieme Verlag, Stuttgart (1958).[6] The apparatus used in the administration of the test consisted of a modified version of the Magnetonphon of the Deutsche Phonak Gesellschaft, Stuttgart.

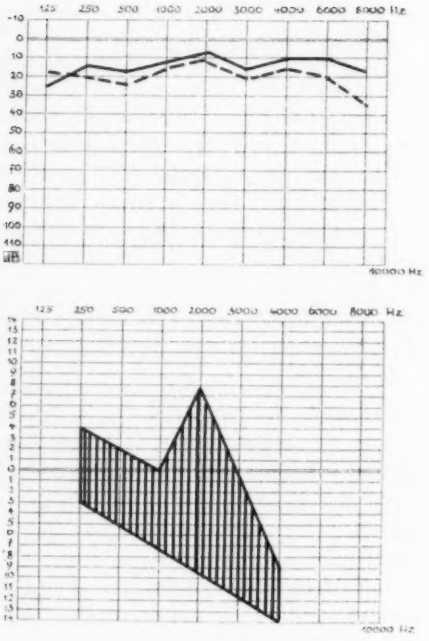


Fig. 4.—Localization band in a woman with an apoplectic hemorrhage in the right subcortical area. Distortion to the left side.

approximately 60 db falling off at higher frequencies: approximately 50 db at 4,000 cps, and even less at 6,000 cps and 8,000 cps.

The test for each signal frequency was started at the maximal interaural time difference of 0.648 msec. By a step-wise lessening of this difference the zone of mid-line impression was gradually established. This is really a zone and not a definite point on the delay-line scale, that is, there remains a final range of uncertainty. This zone is referred to as the "localization band." We usually started delaying the signal to the left ear so that lateralization was first to the right side. When the direction of the delay was reversed it became again necessary to put in a brief pause in order to prevent adaptation and fatigue.

Figure 1 shows the results of this test in normal-hearing subject plotted as frequency vs. a scale from 0 to 14 to either side. This scale represents a tangential function of the interaural time delay, the number one corresponding to the minimal delay of 0.018 msec and the number 14 to the maximal delays of 0.648 msec. The cross-hatched area of Figure 1 shows the zone of mid-line impression: the "lateralization band." With increasing age, this band was found to widen so that in extreme cases, in some senile people, it extended over the complete usable range from number 14 on the right to number 14 on the left. Thus, in addition to the binaural test, the dependence of sound localization upon age, as first described by Matzker and Springborn,[7] represents another quantitative indication of the central fraction of presbycusis. The normal "localization band" for normal-hearing young adults lies always around the zero line of the diagram showing no significant deviation to either side. This is true even for unilateral preponderance, up to 10 db, in cases of hearing losses of peripheral origin. Compensation of the signal sources for unilateral losses have been tried, so far unsuccessfully.

In the following, a few representative test results will be described in which the localization band was found to be pathologically altered.

## REPORT OF CASES

CASE 1 (Fig. 2): Marianne Z., 22 years of age. Audiogram: normal hearing on the right, loss of 15 db for all frequencies on the left. Whispered speech: right 40m, left 4 m. The localization band for all frequencies examined was wide and deviated toward the right side. Upon surgery, a frontal meningioma of 260 gr was found on the left side, i.e., contralaterally with respect to the direction of lateralization.

CASE 2 (Fig. 3): Ilse P., 16 years of age. Threshold audiogram normal, whispered speech normal for both ears. The localization band was normal with the exception of the frequency of 1,000 cps which persistently showed a maximal lateralization toward the right. This was interpreted as indicating pathology in the left cerebral hemisphere or in the right brain stem. Surgery revealed a large temporal meningioma on the left side. This case demonstrates the importance of testing single frequencies. A test with white noise would very likely have produced normal results.

CASE 3 (Fig. 4): Magda S., 34 years of age. For three weeks hemiplegia on the left side. Threshold audiogram normal, whispered speech near normal. The binaural test was clearly indicative of pathology with 26/12/14 mistakes. The lateralization band was considerably widened and deviated toward the left. This finding indicated a pathological process in the right hemisphere. From the neurological examination, a diagnosis was made of an apoplectic hemorrhage in the right subcortical area.

CASE 4 (Fig. 5): Hugo B., 59 years of age. In 1942, patient had been operated for a tumor (?) in the right hemisphere; since that time he had been hemi-

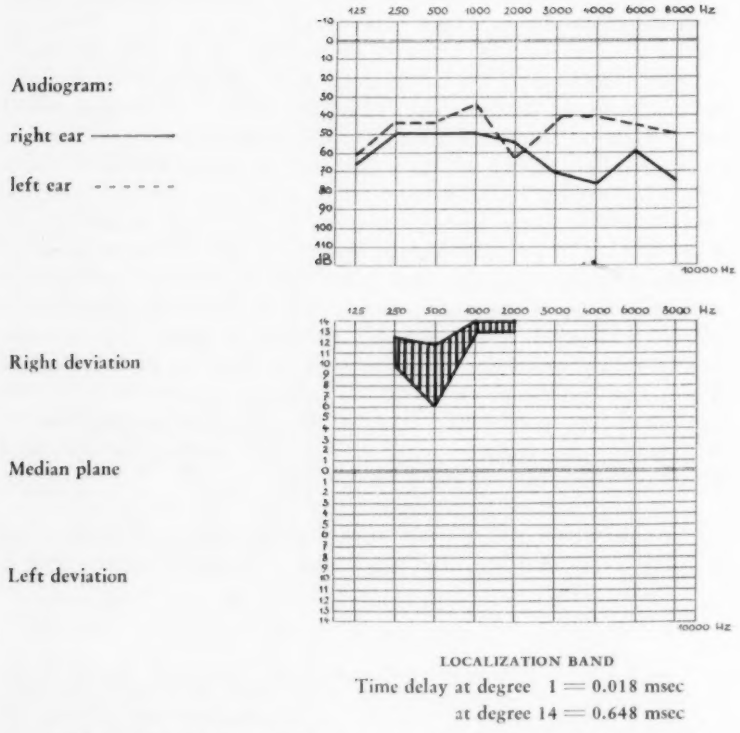


Fig. 5.—Localization band in a man with an obliterated right carotid artery and hemiplegia on the left side. Deviation indicates that the cerebral focus must be situated not only in the right hemisphere, but also in the right brain stem.

plegic on the left. Audiogram: flat loss in both ears. Whispered speech: right 0.4 m; left 0.8 m. Binaural test pathological: 24/11/22 mistakes. The localization band (measured only for those frequencies for which the difference between the two ears was less than 15 db) deviated to the right. Since the cerebral lesion was likely to be on the right side, the results were taken to indicate a functional impairment in the right hemisphere, in the subcortical region as in the brain stem. Arteriography demonstrated a thorotrastoma in the right neck (presumably caused by extra-vascular injection of thorotrast in 1942). The carotid artery was obliterated. Therefore, it appears that the whole right side of the brain was functionally impaired.

CASE 5 (Fig. 6): Herbert S., 46 years of age. Threshold audiogram: bilateral high-tone loss almost symmetrical. Whispered speech: bilaterally 20 m. The binaural test with 41/40/41 was definitely pathological. Localization: the localization band covers almost the entire range from right to left. The only reliable results (2000 cps, deviation toward the left) seems to point to a process in the right hemi-

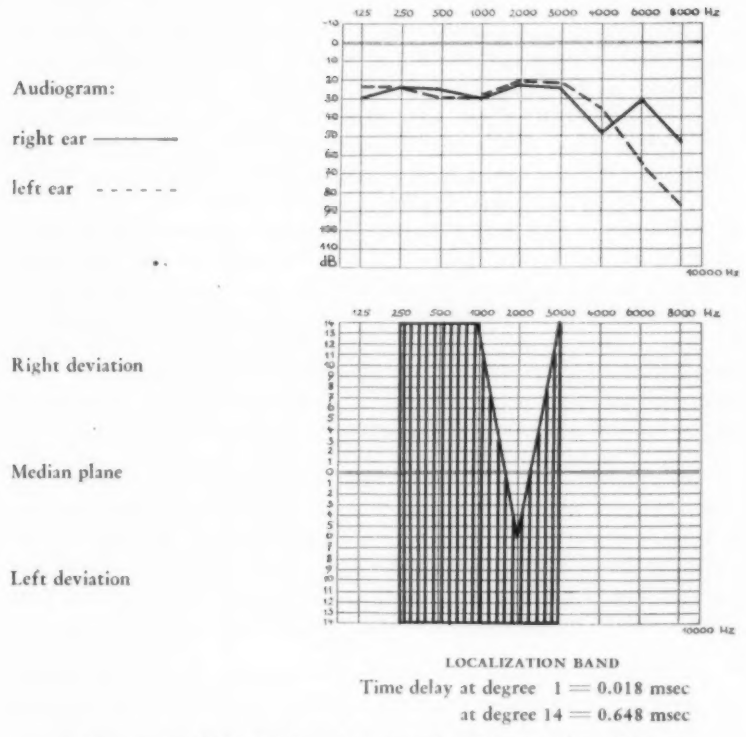


Fig. 6.—Localization band of a man who died some days after examination. Localization function is nearly completely lost. The deviation to the left side at 2000 cycles indicates the focus: autopsy showed a great glio-blastoma in the brain stem on the left side.

sphere or in the left brain stem. The autopsy, after the patient's death, revealed a large glio-blastoma in the left brain stem which appeared to have been inoperable.

One final remark may be in order. Somebody familiar with the quality of the interrupted signal produced by the Peters' audiometer could raise the objection that the audible click occurring with each switching period (cf. above) might have influenced the test results. This is very likely correct. However, since the time constant of these transients did not vary appreciably with frequency according to oscilloscopic observations, it is impossible that the click alone had caused the divergent behavior of single test frequencies as exhibited in Cases 2, 3, and 5. The frequency of the test signal itself must have played a major role in the localization test.

## CONCLUSIONS

Up to the date of this writing, the binaural hearing test (bilateral integration) has been administered to more than 1700 patients and the sound localization test to more than 400 patients, both with encouraging results. These tests appear to provide audiometric tools for the exploration of the central auditory pathways. The test results do not only indicate that a central lesion is present, but, within limits, point to the location of such lesions. While a high score of mistakes in the binaural test is indicative of a functional failure of the synaptic connections within the brain stem, the deviation of the localization band to one side points to a cerebral lesion on the contralateral or to a brain-stem lesion on the homolateral side. It appears that the dividing level above which contralateral localization and below which homolateral localization occurs is the ventral brain stem. In each individual case one has to make sure of course that a deviation observed was not caused by a unilateral peripheral impairment.

In contrast to the results by Sanchez-Longo and by Forster, the present results indicate that the occurrence of abnormal lateralization of sound is not limited to the case of pathological processes in the temporal lobe. When our modification of the testing technique was used processes located in the frontal, parietal, and occipital lobes were also found to contribute to an abnormal localization.

It is hoped that the accumulation of further results will explain the divergent behavior of single frequencies. Additional test methods for the exploration of central auditory functions are now under development in this department.

UNIVERSITY OF MAINZ
MAINZ, GERMANY

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LXXXVI

# CHORDOMA

## STUDY OF A RETROPHARYNGEAL CASE

JACQUES TURCOT, M.D.

J. P. DESPRÉS, M.D.

AND

MARC BEAUDET, M.D.

QUÉBEC, CANADA

Chordomata are apt to grow anywhere along the spine, but with most frequency at its two extremities.

The compilation of statistics by MaBrey,[7] Faust, Gilmore and Mudgett (1944) and Dahlin[3] prove that the sacrococcygeal localization is the most frequent, followed immediately by the sphenoccipital region, below the sella turcica.

Chordoma[5] is a tumor originating from aberrant remnants of the notochord. It is interesting to note that the nuclei pulposi, the real adult tissue (remainder) of notochordal origin seldom gives occurrence to chordomata.

The authors previously cited are unanimous in stating that the chordomata along the spine, excepting the two extremities, occur mostly in the cervical region; and infrequently in the lumbar and dorsal segments. The thoracic chordoma may simulate a mediastinal tumor.[1] In most cases of chordoma, there is some bone destruction,[3] involving one or more vertebrae.

Many authors have emphasized the importance of trauma in the incidence of this tumor; Centil and Coley[4] reporting two occurrences on seven cases and stating that the incidence in the literature is around 30 per cent. Roux[14] reports a case with a traumatic history.

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From the Service of Surgery of l'Hôtel-Dieu de Québec Hospital, Québec, Québec, and from the Department of Surgery, Laval University Medical School, Québec, Québec.

REPORT OF A CASE

Mrs. J. M. H., a white female, 30 years of age, came to the hospital on November 11, 1957, complaining of a progressive nasal obstruction.

This had started about two years previously. There was no discharge. The symptoms had been worse for the last three months; the right naris was completely obstructed and there was some impairment of hearing in the right ear.

There was a large mass in the retropharynx with displacement forward of the retropharyngeal wall. The tumor pushed forward also the soft palate and completely obstructed the nasopharynx. The mucosa of the retropharynx was everywhere normal.

The x-ray picture showed the mass in front of the first and second cervical vertebrae. There was no bone involvement. The superior maxillary bone was normal as were also the paranasal sinuses (Figs. 1 and 2).

The usual laboratory tests were all normal.

A biopsy was performed through the mucosa of the retropharynx on November 29, 1957. A small amount of a gelatinous tissue was removed.

The pathologist's report: chordoma (Figs. 3 and 4).

On December 13, 1957, the tumor was excised. As it was impossible to remove it en bloc through the mouth, it was decided to divide the mandible in the midline and retract both ends laterally to permit a large opening.

The incision, starting at the inferior gingiva in the mouth, was prolonged to the lip and carried on the skin in the midline to the submental region where it followed the left side of the jaw on the mesial border of the bone.

The mandible was cut between two teeth and the floor of the mouth was incised for four or five centimeters to the left side. The duct of the left submaxillary gland was cut and ligated.

It was now felt that the submaxillary gland should be removed. This incision gave a large opening to the palate and pharynx. The

Fig. 1.—Chordoma of the retro-pharyngeal region: lateral view.

Fig. 2.—Chordoma of the retro-pharyngeal region.

soft palate was also divided one-half centimeter on the right side of the midline.

The retropharyngeal space was visualized through a vertical incision of the retropharyngeal mucosa. The tumor was then easily accessible. It was dissected free on both sides and from the vertebrae. On the right side there were seen to be some adhesions to the vertebral aponeurosis. A very thorough curettage of this zone was done after removal of the tumor. There was no hemorrhage or oozing of blood. The retropharyngeal mucosa was closed without drainage. The soft palate was sutured with 00 chromic catgut.

The floor of the mouth was closed with 00 chromic catgut and the bone was approximated with stainless steel wire. The skin was sutured with Dermalon 00000. The immobilization of the mandible was secured by a dental "moulage" prepared in advance.

The operation was concluded with a tracheostomy. The postoperative course was uneventful except for a salivary fistula.

The specimen weighed 25 grams and measured 4x4x3 cm.

The microscopic section showed the characteristic physialiphore cells.

There was a postoperative complication: a salivary fistula of the submaxillary gland on the left side. This gland was irradiated so as to stop its function.

As a curettage of the prevertebral aponeurosis had to be done, it was decided to give an x-ray therapy series to the cervical region; 3500r were administered through two portals.

By August 1958, the patient was well and there was no evidence of recurrence.

In March 1959, the patient was seen again, and there was a recurrence at the same place: the retropharynx. This tumor was excised through the mouth, with longitudinal incision of the soft palate. The early postoperative period has thus far been uneventful.

## COMMENT

Chordomata vary in their location, so their evolution differs. Their progression is slow and the survival period[2,10] without treatment may number many years.

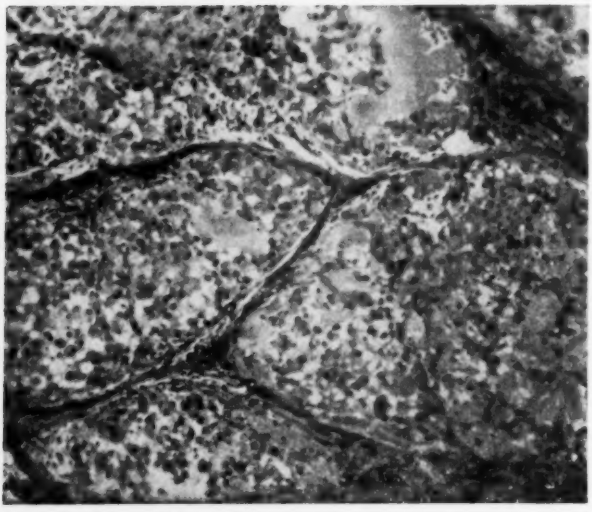

Fig. 3.—Photomicrograph (x180). Chordoma of the retro-pharynx.

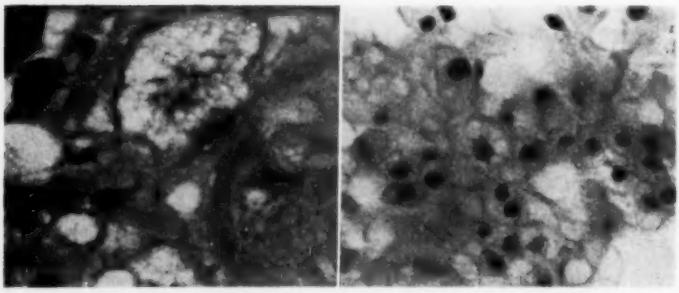

Fig. 4.—Photomicrograph (x720). Chordoma of the retropharynx, showing the physaliferous cells, large globular cells, epithelioid or vacuolar.

This case had a history of two years' duration before the first consultation.

Most of these tumors invade the adjoining bone and give, in their last stages, excruciating pains, by invading or compressing the nerves.

These characteristics of slow progress, few metastases and finally bone invasion are an indication for radical surgical removal, but surgical excision is difficult in most cases.

For this reason, an effort was made to find a good route of approach to tumors of the upper part of the cervical spine.

These wide excisions, with sacrifice of important functional tissue such as nerves, are accepted when there is a likelihood of performing a curative operation but not as palliation, because then it is imperative to give the patient a few years of comfortable life, saving all his important nervous functions.

Usually the slow growing tumors do not respond very well to x-ray therapy. Chordomas are not cured by x-ray[15] therapy, but irradiation is much more useful than it was thought to be few years back. Many examples of arrest of growth, even improvement, have been reported the last few years. We have seen one such case of a sacro-coccygeal tumor. This prompted us to give postoperative x-ray therapy.

It is felt that x-ray therapy may help in the prevention of recurrences,[11] which are so frequent after surgical excision.

## CONCLUSIONS

The surgical excision is the treatment of choice for chordomas.

Description of a route of approach to the upper retropharyngeal region is reported.

Experience indicates that x-ray therapy is of value in slowing down the growth of these tumors and even permitting recalcification of bone.

35, RUE STE-URSULE

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LXXXVII

# RECONSTRUCTION OF THE LARYNGEAL LUMEN IN CHRONIC LARYNGEAL STENOSIS

CHARLES R. SMATHERS, M.D.

JACKSONVILLE, FLA.

Rapid progress has been made in the surgical treatment of chronic laryngeal stenosis since Schmiegelow[1] described his method in 1929. His operation with modifications has given excellent results. There exists, however, a large group of patients who continue to wear a tracheotomy tube because of cicatricial stenosis of the larynx. To emphasize the excellent therapy available, three additional cases of chronic laryngeal stenosis are presented. A discussion of the method employed in the treatment of these cases is also included.

Holinger and Johnston[2] list the following objectives in the management of chronic laryngeal stenosis. "Re-establishment of the normal airway and closure of the tracheostomy; second, the restoration of as serviceable a voice as possible; and third, to provide a larynx that will close satisfactorily to prevent the aspiration of food and fluids during deglutition." Clerf[3] stresses the importance of removal of the obstruction and maintenance of the airway by mechanical means during the healing period. Negus[4] re-emphasized the use of the skin graft as advocated by Arbuckle[5] in the treatment of cicatricial stenosis of the larynx. Other workers[6,7] have found that epithelial lined cavities promote healing and prevent contracture and stenosis. Further modifications in the use of the stent graft in laryngeal stenosis were described by Figi.[8,9] All authors agree that the long standing cicatricial laryngeal stenosis resulting from trauma and inflammatory processes is the most resistant to treatment.

In the management of cicatricial laryngeal stenosis the infectious process must first be removed or controlled before any reconstruction is attempted. Any perichondritis must be allowed to subside prior to undertaking surgical reconstruction of the larynx. The amount of respiratory obstruction as well as the age of the patient are important considerations in selection of candidates. Periodic dilatations may suffice for the mild or moderate degree of stenosis. Many patients refuse to have repeated laryngeal dilatations with metal bougies be-

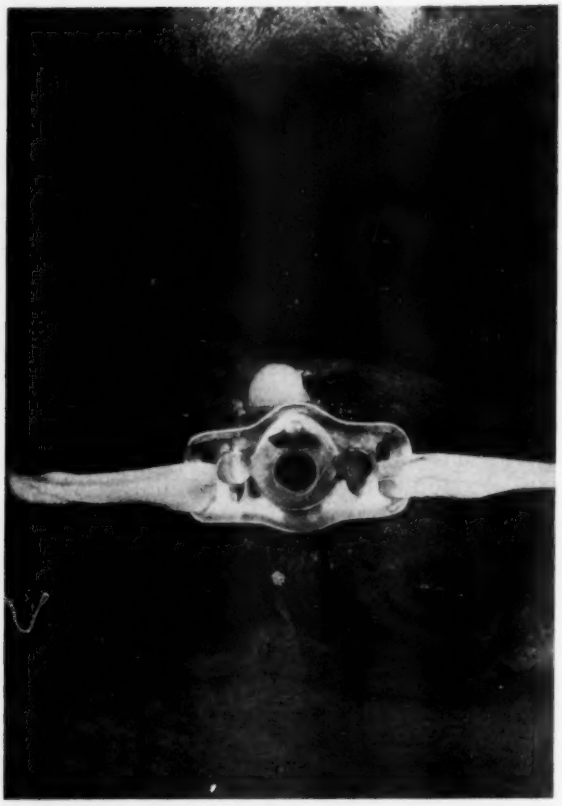

Fig. 1.—Acrylic obturator in position in Case 2. The obturator was worn in this position for six months prior to decannulation. During this period, the tracheal cannula was changed daily by the patient.

cause they are painful. Local anesthesia is not effective in completely removing the stretching sensation. Laryngeal and upper tracheal dilatations in severe cicatricial laryngeal stenosis are not satisfactory because the dilated fibrous tissue quickly returns to its previous position. Complete or partially complete laryngeal stenosis, in which a permanent tracheotomy is present, requires excision of the stenotic area and surgical reconstruction of the laryngeal stenosis. The growth of the larynx in chronic laryngeal stenosis as cited by Jackson[10,11] has not been a factor in the cases presented below.

Before any surgical reconstruction can be undertaken, a low tracheotomy should be performed away from the stenotic area. High tracheotomy with damage to the supporting cricoid cartilage is one of the most common causes of laryngeal stenosis. The low position of the tracheotomy must be maintained in a relatively normal portion of the trachea where no narrowing of the lumen exists. Any tendency for the cannula to progress upward again to the stenotic area must be avoided. Holinger[2] cautions against this complication and advises passing the tracheotomy tube tapes beneath the arms in difficult cases.

Reconstruction of the laryngeal lumen is a three-stage procedure requiring 6 to 12 months for completion. A laryngostomy approach is employed. The first stage is removal of the stenotic area and insertion of the skin graft. Constant pressure on the skin graft in the area of removal of the stenosis is obtained by the use of a foam rubber stent. This is secured in position by through-and-through external steel wire suture above the tracheotomy tube.

The second stage is performed 8 to 10 days later with removal of the rubber stent and insertion of an acrylic obturator. The exact size of the obturator is determined by making a pattern of the new tracheal and laryngeal lumen with dental compound. Two obturator patterns are made, one remaining in the larynx while an acrylic duplicate is made from the other. When the acrylic obturator has been made, the dental compound mold is removed and the acrylic obturator substituted. The acrylic material is light, durable and inert, suitable for prolonged wear. By the use of the pattern mold, the correct fit is therefore assured. The obturator prevents any contracture of the tracheal and laryngeal lumen during the healing period.

The third stage involves removal of the acrylic obturator, closure of the laryngeal stoma, and decannulation. This stage is performed 6 to 9 months after the obturator has been inserted into the tracheal and laryngeal lumen. The third stage is performed only when the new lumen in the larynx and trachea remains open without the obturator for a period of two weeks. All scar contracture has occurred and the lumen will be maintained without the stent.

The following three cases are presented as examples of management of chronic laryngeal stenosis, together with complications and experiences encountered in their successful conclusion. All cases were from the Bronchoscopy Department of the Manhattan Eye, Ear and Throat Hospital and were managed under the direction of Doctors William Rabbett and Daniel C. Baker, Jr.

REPORT OF CASES

CASE 1. K.Z., a 60 year old white female with a history of diphtheria and scarlet fever in 1903 at the age of eight requiring emergency tracheotomy. In 1905 emergency tracheotomy was again necessary because of recurring "proud flesh" obstructing the laryngeal airway. In 1910 at the age of 15, the patient again had difficulty with breathing because of the recurring granulation tissue in the larynx. Removal of the obstructing tissue was performed by direct laryngoscopy and a Mosher life-saving tube was inserted postoperatively. The patient did not tolerate the Mosher tube and prompt removal was necessary. After removal, however, the laryngeal and tracheal lumen remained adequate, and the patient had no difficulty for a period of 23 years. Tracheotomy was again performed in 1933 because of recurrence of infected granulation tissue in the larynx obstructing the airway. Evidence of tracheal stricture by fibrous tissue was recorded at laryngoscopy one week later. Subsequent decannulation postoperatively was effective for another 21 years. The patient was first seen in 1954 in the Bronchoscopy Department of the Manhattan Eye, Ear and Throat Hospital with acute laryngotracheitis causing respiratory distress. Tracheotomy was necessary because of the edema and swelling of the larynx in addition to the pre-existing subglottic stenosis. Planograms of the larynx on this admission revealed "narrowing of the subglottic larynx and upper trachea. The laryngeal ventricles are not visualized." The remainder of the laboratory studies were within normal limits. Examination of the larynx when the edema had subsided revealed a normal epiglottis; phonation and respiratory movements were made with the thickened ventricular bands. The true vocal cords were fused with the ventricular bands obliterating the laryngeal ventricles. Limited mobility was present bilaterally. Subglottic narrowing of the laryngeal and upper tracheal lumen with dense scar tissue was present.

Subsequent repeated attempts at laryngeal dilatation and decannulation were a failure. In December 1955, laryngostomy and excision of the stenosis was performed with insertion of an acrylic obturator with a ring as described by Goodyear and modified by Figi. No skin graft or foam rubber stent was used. Two weeks later the obturator was removed by direct laryngoscopy. The patient was discharged with the tracheotomy tube. Periodic follow-up laryngeal and tracheal dilatations resulted in successful decannulation five months later. The patient now requires infrequent laryngeal and tracheal dilatations, the most recent in October, 1957. Her voice is of a husky quality and she has had no aspiration difficulties with

deglutition. However, the airway is adequate only for moderate activity, and the patient has occasional dyspnea on exertion.

*Comment.* The prolonged history of repeated laryngeal trauma at an early age was the contributing etiological factor in this case. Laryngeal dilatations were a failure. A more adequate postoperative tracheal and laryngeal lumen may have been obtained if a skin graft had been employed.

CASE 2. C.E., a 48 year old colored female with a history of progressive breathing difficulty since 1944. Her history revealed that two thyroid operations had been performed, the first in 1920, the second in 1927. Examination on admission in 1954 revealed an infantile epiglottis with complete fixation of the left vocal cord in the midline. The right vocal cord had only limited movement. The rima glottidis was a narrow slit. The patient was very obese (240 pounds) and had diabetes mellitus. Serology was negative. The remainder of the physical examination and laboratory studies were within normal limits. A low tracheotomy was performed on this admission.

Right arytenoidectomy performed one month later was unsuccessful because of postoperative scarring resulting in laryngeal stenosis. In November, 1955, a laryngostomy and excision of the stenotic fibrous tissue was performed with insertion of an acrylic obturator with a ring as described by Goodyear and modified by Figi. Five weeks later the obturator was removed by direct laryngoscopy, and the patient was discharged with the tracheotomy tube. Weekly laryngeal and tracheal dilatations for three months were not effective for decannulation. In October, 1956, a recurrence of the colloid goiter was removed because of suspicion of malignant change. The patient was decannulated before discharge, only to be re-admitted January 15, 1957, for tracheotomy because of respiratory distress.

In June, 1957, surgical reconstruction of the laryngeal lumen was started with excision of the stenotic fibrous tissue and insertion of a skin graft. The foam rubber stent and graft were coughed out of the larynx four days postoperatively. The patient was discharged and followed with periodic dilatations of the tracheal and laryngeal lumen. Decannulation was unsuccessful.

Surgical reconstruction of the laryngeal lumen was again started in December, 1957. The firm, fibrous stenosis was excised and a split thickness skin graft was inserted around a foam rubber stent. Eleven

Fig. 2.—Acrylic obturator employed in Case 3. The lateral view is shown above and the top view below. This solid obturator remained in the larynx and upper trachea for seven months before decannulation.

days later the stent was removed and the obturator pattern was made from dental compound material. Ten days later the permanent acrylic obturator was inserted through the laryngeal stoma in the neck above the tracheotomy tube. The obturator remained in position for six months. The new laryngeal and tracheal lumen was maintained when the obturator was removed. The patient was decannulated in July, 1958. A plastic closure of the laryngeal stoma with excision of the scar tissue was done in August, 1958. The patient has a husky voice and no swallowing difficulty. The airway is adequate only for moderate activity, and dyspnea is present on exertion.

*Comment.* Bilateral abductor paralysis was the initial cause for this patient's symptoms. Operative laryngeal trauma contributed to the establishment of the stenosis. Laryngeal dilatations were a failure. Arytenoidectomy was a failure. The first stage of surgical reconstruction of the laryngeal lumen failed because the patient coughed out the skin graft and stent. Obesity and diabetes complicated the management. The overweight contributes to the dyspnea present on exertion.

CASE 3. J.P., a 32 year old white male with a history of chronic laryngeal stenosis of 15 years' duration. His stenosis followed an attack of laryngotracheobronchitis in 1943, requiring tracheotomy. Following the subsidence of the inflammatory process, repeated dilatations of the stenotic area were not successful. The trauma of dilatations resulted in a severe inflammatory reaction and granulation tissue formation. In an effort to diminish the granulation tissue, x-ray therapy was given over a two month period at weekly intervals; 900 roentgens, air dose, was given to each side of the neck, total 1800 R, 200 kv machine. Irradiation had no effect on the stenosis. The tracheal and laryngeal lumen was still not adequate for decannulation.

The patient was not seen again until February 1958. Examination of the larynx at that time revealed free movement of the right vocal cord, fixation of the left cord, and left subglottic fibrous stenosis blocking the airway. The tracheotomy tube had been worn since 1943. Planograms of the larynx confirmed the subglottic stenosis on the left. The remainder of the laboratory studies and physical examination were within normal limits. The reconstruction of the laryngeal lumen was started in March, 1958. A laryngostomy was performed and the stenotic area was excised. A split-thickness skin graft was inserted over a foam rubber stent. This was secured in position by external steel wire sutures, anchored to the exposed cartilage. Ten days later the stent was removed and the obturator pattern made from dental compound material. The next day the permanent acrylic obturator was inserted through the laryngeal stoma in the neck above the tracheotomy tube. The obturator remained in position for seven months. When the acrylic obturator was removed, the patient had some swallowing difficulty but this was corrected by tube feeding for several days. The tracheal and laryngeal lumen was maintained, therefore the laryngeal fistula was closed and the patient decannulated in November, 1958. At present, the airway is adequate. The voice is weak, but is becoming stronger with use. No aspiration difficulties are present.

*Comment.* A severe laryngeal and tracheal infection prior to antibiotics was responsible for the subglottic scarring and stenosis. X-ray therapy was of no value except to diminish the infection and granulation tissue in the larynx. The age of the patient was favorable to the reconstruction. A short period of retraining of the swallowing mechanism was necessary after removal of the acrylic obturator. A more adequate airway in this case resulted in a poorer voice quality.

SUMMARY

Three cases of laryngeal stenosis are presented with comments on their management in surgical reconstruction of the laryngeal lumen. The method employed consisted of three stages requiring 6 to 12 months for completion. Each patient had worn a tracheotomy tube for a period of 1½, 3 and 15 years respectively prior to surgical reconstruction of the larynx. In each instance it was possible to reconstruct the laryngeal lumen by an external approach with a functional result.

1842 KING STREET

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# Abstracts of Current Articles

## EAR

### Response of the Organ of Corti to Ultrasound

*Beck, Chl.*: Arch. Ohr-, Nas.-, u. Kehlk.- Heilk. 174:173-181, 1958.

The author applied ultrasound of varying doses directly to the cochlear capsule in guinea pigs. Small dosages ($3W/cm^2$) resulted in reversible changes: pyknosis of haircells and decrease of ribonucleic acid in the cell plasma. Larger dosages ($6W/cm^2$) produced damage of all cochlear structures progressing with time after exposure. Thirteen days after application, the organ of Corti had collapsed over large stretches; the outer haircells were missing. In addition, the osseous capsule showed broadening of the enchondral layer as well as vacuolization and proliferation of the endosteum. Comparing these results to those seen after local application of heat in earlier experiments of the same author, he concluded that the effect of ultrasound upon the cochlea is largely a mechanical, vibratory one.

TONNDORF

### The Use of Polyethylene Tubing in Tympanoplasty

*Stapff, V. H.*, and *Berton, S. A.*: Anales de Oto-rino-laringologia Del Uruguay 29: Part I, 1959.

The authors of this article advocate the use of polyethylene tubing, i.e. (PE 20 Clay Adams), in tympanoplasty. The tube is put into place via the middle ear during the operation: it emerges through the nasal fossa, and is fixed at that level. The purposes of the described tubing are as follows:

1. Serves as a continuous suction device during the operation.

2. Serves as a postoperative drain, carrying fluids (specially plasma) from the middle ear outwards.

3. The most important purposes, the author believes, are:

   a) as a way for easy, controlled, sterile, postoperative insufflation of the created middle ear cavity.

   b) the possibility of introducing easily, solutions of different kinds into the same cavity.

   c) as an auxiliary device for holding the graft in its correct position away from the middle ear mucosa, thus avoiding adhesions.

Polyethylene tubing has remained in place for an average of 21 days in most of the cases. In one patient the tube had remained in place for about ten months, with no trouble whatsoever. In some cases they have left the tubes permanently and in these types of cases they have used the polyethylene tube PE, 190.

OBREGON

**Permanence of Hearing Results Ten to Seventeen Years After Fenestration for Otosclerosis: Including Case Report of Closure of a Fenestra Seven and One-Half Years Atfer Successful Fenestration**

*Hilleman, Geo. A.*, and *Shambaugh, Geo. E. Jr.*: A. M. A. Arch. Otolaryng. 69: 136-142 (Feb.) 1959.

A review of the permanence of fenestration results seems timely in view of the present popularity of the stapes mobilization procedure.

Four previous long-term studies of fenestration results (Adin and Shambaugh, House, Hall——all in 1951, and Campbell—1954) on series of patients varying from 150 to 390 in number and covering periods of observation from 2 to 3 years up to 5 to 10 years, are reviewed. Criteria for judging results were not defined in all instances but successful results appeared to range from 56% to 82% of a series (or fraction of a series) reported.

The authors report to 10 to 17-year postoperative hearing results in 690 fenestration operations (including one patient whose successful fenestration closed after 7½ years of maintained practical hearing). They reviewed 1743 fenestration operations performed between August 1939 and January 1948. Of these 1743 cases, 690 (40%) returned 10 or more years (10 years to 17 years 10 months) after operation for audiograms. The surgical technique employed during

these years represented a progressive evolution from the original Lempert technique to an improved technique described by Shambaugh and Juers in 1946. The latter technique, paying particular attention to factors that influence osteogenesis, has continued in use with few changes since 1946.

Definition of terminology used:

*Criteria for Designating Results*

30 db level of "practical" hearing in most recent audiogram . "successful" result

Average level of 31 db or more for speech frequencies . . "unsuccessful"

Gain of 11 db or more for speech frequencies . "significant" improvement

Loss of 11 db or more . . "significant" loss

Hearing within 10 db of pre-op. level . . "unchanged by surgery"

A suitability—normal preoperative cochlear function for speech frequencies

B suitability—one of the speech frequencies impaired below normal range by bone

C suitability—two or all three speech frequencies impaired below normal range by bone

The final level of hearing at the most recent test in these 690 fenestration operations 10 to 17 years after surgery showed that 43% remained at the 30 db level (successful), 57% had a loss of 31 db or more for the speech frequencies (failures). As anticipated, the percentage of successes in A suitability was 56, B 27%, and C 8%. Stabilization and maintenance of maximum improvement in the operated ear occurred in 61% of the 690 cases, 26% experienced a partial loss of maximum improvement, 3% failed to obtain improvement, and in 2% the hearing became worse. Of the 8% who lost all of the improvement over the years, 1/3 resulted from neurosensory deterioration, while two-thirds probably were caused by closure of the fen-

estra by bone and fibrous tissue—an over-all percentage of 5% of closures. The rarity of fenestral closure later than 2 years postoperatively is emphasized by the fact that only 2 such cases were encountered in this series of 690 followed for 10 to 17 years. The authors and their associates performed more than 4300 fenestration operations during the past 20 years, and only two cases of proved closure of the fenestra occurred later than 2 years after operation—one case, which they previous reported, that closed between the third and fourth year, and the case reported in the current study, that closed between 7 and 9 years after operation.

The dependability and permanence of hearing improvement in the majority of fenestration operations performed by modern techniques is apparent.

HILDING

**Histopathological Studies of Soft and Bone Tissue in the Temporal Bone of Acute and Chronic Otitis Media**

*Iwatake, Toyohara*: Japanese J. Oto-laryng. 62:1541-1567 (July) 1959.

Iwatake recorded his observations on the tissue studies from eight acute and 60 chronic otitis media and chronic mastoiditis cases. The ages were 1 to 60 years.

1. Changes in the soft tissues. In mild cases of acute and chronic ears, the epithelial layer was preserved but the cells became tall. In the severe cases these cells had disappeared. Subepithelial and periosteal layers were edematous. There were hemorrhage, congestion, cellular infiltration, and proliferation of fibroblasts of various grades. Occasionally cells contained serous and purulent exudate.

2. Changes in bone tissue. In the areas with mild inflammatory reaction, the nuclei of osteocytes were pyknotic. In the areas with severe reaction, the nuclei disappeared. Bone lamellae were indistinct and faintly stained with hematoxylin. Where the reaction was most severe the bone tissue failed to take the stain due to halisteresis. Cellular infiltration, congestion, hemorrhage, proliferation of fibroblasts and fibrocytes was noted in the marrow space.

Widening of Havers' canals and inflammatory changes in the widened Havers' canals were also seen. At the edge of the bone tissue,

regeneration by osteoblasts and lacunar resorption by osteoclasts was observed. In all the 8 acute cases osteitis was found.

In the chronic cases at the edge of the bone tissue, there were osteoblasts, newly regenerated bone, sometimes osteoid tissue, osteoclasts, lacunar absorption, new bone tissues, the necrotic bone cavities, and occasionally there were boundary lines of new and old bone which were darkly stained by hematoxylin. In severe and long continued infection, all systems of the temporal bone had almost completely disappeared, except in the area near the tympanic cavity and the antrum.

Iwatake holds the view that such a histopathologic study reveals that the mastoid process and petrous portion of the temporal bone undergo a similar change in the tissue reaction—though in different degrees. For this reason, Iwatake names the mild acute otitis media pneumatocellulitis temporalis acuta, and the severe ones osteitis or osteomyelitis temporalis acuta. All cases of chronic otitis media are histopathologically osteitis or osteomyelitis temporalis chronica.

3. Comparison of acute cases with chronic otitis media. Changes in both soft and bone tissue in acute cases are much simpler than those in the chronic cases. In chronic cases the process of the regeneration and absorption occurs simultaneously in various portions of the diseased bone.

HARA

**Observation on the Healing Process of Operated Temporal Bone of Chronic Otitis Media**

*Nishihata, H.*: J. of Oto-Rhino-Laryng. Soc. Japan 62:1295-1325 (June) 1959.

Nishihata recorded his histopathological observations in 122 cases of chronic otitis media which came to the Keio University Clinic in which radical mastoidectomy was performed.

Complete wound healing was obtained in 62.3% of the total number operated. Among those with incomplete wound healing, the difficulty was seen in the window niche in 37%; at the tubal orifice in 28.2%; in the hypotympanum in 23.9%; on the promontary in 8.7%; in the sino-dural angle in 8.7%; in the retrofacial area in 6.5%; in the attic 4.3%, and in the perisinous area in 2.2%.

Forty-nine cases had revisions. Incompletely removed mastoid cells and mucosa were found in most of the cases.

In 15, incomplete healing occurred, though at first the operation appeared to be satisfactory. Microscopic examination revealed that failure of healing was due to the presence of infection in the mucosa which interfered with proper epidermatization. Morphologically, the most conspicuous change was the infolding of the mucosa and a cyst formation. The cyst has also been observed beneath the epidermis where the clinical healing had been satisfactory for a period of time.

At the orifice of the eustachian tube there is an extension of the tubal mucosa into the operated area. Where the remnant of the middle ear mucosa and the tubal mucosa come in contact, there was a tendency for development of cysts and formation of an air cavity. Diseased bone did not always interrupt the epidermization. The text contains 23 photomicrographs, 15 diagrams and 57 references.

HARA

**Screening Test for the Deafness**

*Horiguchi, S., Moriyama, S., Idemithu, M., Ebihara, I., Harigaya, M., Ikeda, H.,* and *Nozaki, T.*: J. Otol. Society 62:1411-1418 (July) 1959.

Horiguchi and associates report their experiences in a simple screening test for the early diagnosis of occupational deafness, streptomycin deafness, and deafness in school children. Each of these three types of deafness is known to present a characteristic audiogram. Their procedure was found to be accurate and less time consuming.

1. Occupational deafness in 1128 persons. A pure tone of 4000 cps at 30 db was selected. The screening error was 1.55% and 1.91% in each ear, and this was attributed to the tinnitus.

2. Streptomycin deafness in 1045 persons. Eight thousand cps was tested at 15 db without error. Since there is no known cure for an advanced case of streptomycin deafness, the best preventive therapy is early detection of deafness caused by this drug. Toxicity of this drug appears first in the highest pitch. They selected 8000 cps, and testing one ear was sufficient to meet the object of examination. There were 185 patients who were screened out.

3. Deafness in 5382 school children. Two pure tones of 1000 cps and 4000 cps at 20 db were used; the results compared with the previous mass testing of a large number of school children using 500, 1000, 2000 and 4000 cps at various thresholds.

The authors state that the present method is rapid and less tiresome for the examiners and the examined; hence was found to be most practical. In each of these three groups they were able to test 80 to 150 persons in an hour.

HARA

## NOSE

**Nasopharyngeal Malignant Tumors in Surabaja and Vicinity (Indonesia): Preliminary Report**

*Djojopranoto, Moeljono,* and *Marchetta, Frank C.*: A.M.A. Arch. Otolaryng. 69: 155-159 (Feb.) 1959.

For 25 years the racial susceptibility of the Chinese to nasopharyngeal carcinoma has been known. One author reported in 1951 that it was the second commonest variety of malignant disease in Hong Kong and that it represented 18% of all malignancies. Others noted in 1954 that the incidence of nasopharyngeal malignancy in Singapore was 16.14% of all malignancy. Contrasting with the Chinese incidence, a Swedish study in 1947 reported an incidence of only 0.4% of nasopharyngeal carcinoma among all cancers occurring in Scandinavia. A 1957 Canadian study showed its incidence there to be 0.3%. Other writers doubted that this predilection for nasopharyngeal carcinoma extended to other Oriental races and studies in India and in Japan showed it to be rare among the Indians and the Japanese. Figures indicating the incidence of nasopharyngeal carcinoma among Indonesian natives are not available in the literature but the authors found the incidence to be high in Indonesian natives.

At the Department of Pathology of the Surabaja Medical School in Indonesia they studied 160 cases of nasopharyngeal cancer occurring between the years 1952 and 1955. The majority of these malignancies were epidermoid carcinomas with varying degrees of differentiation. Racial incidence analysis showed 107 among Indonesians, 47 among Chinese and 6 among Caucasians. In the Indonesians, the ratio of men to women so afflicted was 2:1. Cancer of the nasopharynx was

found to be one of the commoner malignancies of the Indonesians and Chinese in Surabaja and vicinity during this period.

Another investigator found a high incidence of malignant tumors of the lymph nodes in the neck in Indonesia. The authors believe that many of the so-called "primary cervical reticulum cell sarcomas or reticulo-endotheliomas" described by him were actually metastases from carcinoma of the nasopharynx. They caution that a diagnosis of a primary cervical malignant lesion of the neck must not be made until the nasopharynx has been thoroughly examined.

HILDING

**A Case of Carcinoma of Maxillary Sinus in a Twelve Year Old Boy**

*Goto, T.*, and *Murashima, J.*: J. Otolaryng. Soc. Japan 62:1292-1294 (June) 1959.

Goto and his associate recorded a case of basal cell carcinoma of the left antrum in a 12 year old boy.

Preoperative radiation of 3150 R was given. Four days later a radical neck dissection and excision of the left superior maxilla followed under intratracheal anesthesia. The postoperative course was smooth, and two years following the operation there has been no recurrence.

Carcinoma of the superior maxilla under 20 years of age is rare. Only six cases in this age group have been recorded in the Japanese literature. Among 1011 cases of maxillary cancer collected in the world literature, only three were under 20 years of age.

HARA

## THROAT

**Management of Dysphagia Due to Retained Meat in the Esophagus**

*Brick, Irving B. L.*: A.M.A. Arch. Otolaryng. 69:151-154 (Feb.) 1959.

In the past four years the author has cared for four patients suffering from dysphagia due to large particles of meat lodging in the upper end of the esophagus. These patients were primarily elderly

with edentia or dentures, onset of their symptoms usually occurred immediately after eating meat, all indicated the area of the supraclavicular notch as the site of obstruction, the drinking of liquids did not relieve the obstruction, and none was able to swallow solids. An esophagogram was obtained in each instance; no intrinsic esophageal disease was noted in any. Esophagoscopy was done in the first three cases and in no instance was the meat particle seen, but each of the three patients obtained immediate and permanent relief of symptoms following esophagoscopy. It was concluded that the meat particle must have been dislodged by the preliminary passage of the obturator into the upper esophagus and the meat particles had passed down into the stomach. (The reason for obstruction in the upper portion, in an apparently normal esophagus, is not clear.) Thus the esophagoscopy itself did not seem essential to the relief of symptoms and the next (fourth) patient was treated by passage of a rubber tube of adequate size (30 F) with equally good results.

The author observed a fifth case, subjected elsewhere to forceps removal of a retained meat particle during esophagoscopy; death ensued from sequelae of esophageal perforation which occurred during instrumentation.

These observations led Brick to think that a harmless and simple means of dislodging meat, and probably also vegetable substances, in the esophagus could be accomplished by the passage of a large rubber tube, after a preliminary esophagogram, but without preliminary esophagoscopy. He warned that evidences of avitaminosis should be looked for prior to passage of the tube, in the even this condition might make the esophageal wall more susceptible to rupture. Removal of metallic objects and bones by this method was not advocated.

HILDING

## MISCELLANEOUS

### Animal Experiments on the Origin of Cholesteatoma in Skin Transplants; Comparison of Fresh Autoplastic and Preserved Homoioplastic Material

*Burian, K.*, and *Cancura, W.*: Zschr. Laryng. 38:577-584, 1959.

Occurrence of "cholesteatom"-like growth in skin transplants used in tympanoplasty has been reported earlier (cf. abstract Beikert, this journal 67:1222, 1958). The present authors examined the fate

of skin transplants in experimental animals (rats), using: 1) fresh autotransplants, 2) fresh homoiotransplants, 3) preserved homoiotransplants; method of preservation: storage in "Thyrode" solution for a period of eight days in 4° C. Transplants were biopsied 10, 18, 28, and 56 days after surgery.

In all cases the original epithelium of the transplant was found to be sloughed off almost totally and to be replaced by newly generated epithelium. In groups 1 and 2, there was marked tendency to gross proliferation during the period of regeneration. Processes of epidermis grew into the deeper cuticular layers. Later on some of these processes became separated from the surface layer showing an ultimate development greatly resembling a typical cholesteatoma. In the preserved material (group 3), the regeneration of epidermis was largely confined to the formation of a smooth surface layer. Growth into the deeper cuticular layers was seen occasionally, but much less frequent than in the two other groups. Consequently, development of cholesteatoma was seldom observed in group 3 (preserved transplants).

TONNDORF

**ACTH and Cortisone in Otorhinolaryngology**

*Bartual, Rafael*: Acta Oto-rino-laringologica Ibero-Americana, No. 3, 1959.

In this article, the function of the pituitary gland and the suprarenal glands is quickly reviewed. The relationship between ACTH and cortisone is briefy summarized, and the influence of these substances in different processes, such as inflammation, allergy, etc., are reviewed.

The action of the steroids in the different otologic, rhinopharyngeal, nasal, paranasal, and laryngeal conditions are well outlined in this article.

In otology, the author believes that steroids are mainly used topically. Ointments containing steroids are of great help in cases of external otitis of allergic origin. When the patient has traumatized the ear, and a superimposed infection is present, the author agrees with Senturia that the addition of Neomycin® and Chloromycetin® to one of the topical steroids is of great benefit to these patients.

The author believes that in external otitis, caused by swimming, with formation of exudate in the ear canals, simple administration of a combination of alcohol and acetic acid (3%) will usually solve the problem, and steroids are rarely indicated in these cases. Patients with chronic otitis media and central perforations, benefit a great deal by the combination of steroids with antibiotics, and steroids are also useful in patients with mastoid cavities. The authors are now trying the administration of steroids following tympanic stapes mobilizations, and are comparing their results with the patients in which this substance was not used.

The author also advocates the use of steroids by mouth in certain cases of eustachian tube salpingitis caused by hyperplasia of lymphoid tissue along the eustachian tubes, cases in which allergy is strongly supected.

Steroids are also used in cases of allergic rhinitis and sinusitis, in cases of laryngeal edema of an allergic nature, and also in patients with lye burns of the esophagus.

OBREGON

**Language Development Among Survivors of Premature Birth**

*Kastein, Shulamith,* and *Fowler, Edmund P. Jr.*: A.M.A. Arch. Otolaryng. 69:131-135 (Feb.) 1959.

The number of children with communication disorders is steadily increasing in this era in which medical, surgical and therapeutic advances are saving so many infants' lives but not always preventing damage to their central nervous systems. A large number of these children represent premature births.

The authors point out that language depends upon the structural, psychomotor, intellectual and emotional integrity of the organism and thus a study of language development can serve as an additional differential diagnostic aid and prognostic tool in determining areas of dysfunction not otherwise discernible at an early age. If delayed or inadequate communication is found, a search for its cause may facilitate diagnosis, and enables undertaking habilitative steps at an age (three to four years) when they are most effective. Absent or defective functions of perception may, at that age, be developed by training, which will not only aid in developing speech, but will stimulate

growth in other areas as well. If this is accomplished, further deviations, which might interfere with learning ability at school-age, may be prevented. Serious problems which might appear at a more advanced age may be prevented by early language and speech evaluation.

The authors report a study evaluating language and speech development in 66 survivors of premature birth at two years of age. The method of evaluation was such that it cut across the entire developmental profile of the child and emphasis was placed upon evidence of central nervous system dysfunction. The method of observing responses to stimuli, symbol behavior, perception, comprehension of speech, contacts, and communication is described.

Analyzing the data made available by these observations, the authors found that 37 of the 66 children had retarded development of language, with or without central nervous system deficit, while 13 of the children with adequate or advanced language development also showed evidence of central nervous system involvement in their responses or behavior during the examination. These data confirmed the validity of language evaluation in young children and its use as an additional tool in differential diagnosis in an attempt to determine areas of dysfunction which might otherwise be overlooked at an early age.

HILDING

# Books Received

**Surgery of the Ear**

By *George E. Shambaugh, Jr., M.D.*, Professor of Otolaryngology, Northwestern University Medical School. Cloth, 669 pages 8 x 11, 326 illustrations, some in color. Philadelphia, London, W. B. Saunders Co. Price $27.50.

One of the most thoroughly satisfying texts which has come to this desk in many a year is this volume by Dr. George Shambaugh. In a definitive exposition the material is presented without bias, a thing difficult to achieve in dealiing with a subject over which there has recently been so much controversy.

The first 145 pages are devoted to fundamentals: embryology, anatomy, the basic elements of diagnosis, and the principles of temporal bone surgery. The three sections which follow comprise the Surgery of Infections of the Ear, the Surgery of Deafness and the Surgery of the Facial Nerve, Endolymphatic Hydrops and Tumors of the Ear.

Descriptions of operative procedures are written with the utmost clarity of detail and the illustrative material is copious and of the highest order. The approach is scholarly and sympathetic and there is a background of historical development in contrast to the sketchy references which often characterize works on contemporary surgery.

The student will derive much pleasure from the collection of some twenty portraits of the pathfinders in otology, past and present, many of whom have hitherto been only names to him, and now come to life in their proper perspective.

Typography and format are in keeping with the quality of the text.

**Peripheral Facial Palsy: Pathology and Surgery**

By *Karsten Kettel, M.D.*, Chief Surgeon, Department of Oto-Rhino-Laryngology, Frederiksborg Central Hospital, Hillerod, Denmark. Foreword by *Terence Cawthorne, F.R.C.S.* 341 pages, 127 figures (12 color plates). Copenhagen, Ejnar Munksgaard. Springfield, Charles C. Thomas, 1959.

This is a very interesting and readable account of the causes and treatment of peripheral facial palsy. Dr. Kettel draws upon a very large personal experience and extensive knowledge of the literature. As mentioned by Mr. Cawthorne in the foreword, a refreshing feature of the book is the objective manner in which the author examines and criticizes the management of his own patients.

The monograph is well illustrated and contains several beautiful color photographs of exposed facial nerves. The bibliography contains a most comprehensevie review of the literature and throughout the book, generous credit is given to those who contributed.

Dr. Kettel's book is recommended to all who contemplate otologic surgery.

S.

**The Surgical Treatment of Facial Injuries**

By *Varaztad Hovhannes Kazanjian, C.M.G., D.M.D., M.D., F.A.C.S., D.Sc. (Hon.)*, Professor Emeritus of Plastic Surgery, Harvard University; Plastic Surgeon Senior Active Staff, New England Deaconess Hospital, etc., and *John Marquis Converse, M.D., F.A.C.S., Lawrence D. Bell*, Professor of Plastic Surgery, New York University College of Medicine; Director of the Institute of Reconstructive Plastic Surgery, etc. (Second Edition) Cloth, large 8vo., 1113 pages, 1155 ills. Baltimore, The Williams and Wilkins Co., 1959. Price $22.00.

An impressive volume by two outstanding authorities. As in the previous edition, which appeared ten years ago, the authors have confined their presentations to techniques with which they have had personal experience; but this experience, it should be stated, is so extensive and comprehensive as to satisfy most readers with its scope.

They have had the collaboration of nine colleagues in some of the more specialized aspects of the subject. Space does not permit their enumeration nor a very useful discussion of the many details covered in the book. Let the comment suffice that the work is authoritative in all respects and presented with the completeness and clarity which characterized the first edition.

In addition to the customary revisions the present book contains six new chapters: on the fractures of facial bones; of the frontoethmoidal region and traumatic cerebrospinal rhinorrhea; facial injuries in children; roentgen examination of the facial bones; the transplantation of tissues; and scars of the face.

# Notices

## AMERICAN OTOLOGICAL SOCIETY

The American Otological Society will hold its annual meeting at the Deauville Hotel, Miami Beach, Florida, March 13-14, 1960.

PROGRAM

SUNDAY MORNING, MARCH 13, 1960

9:00 A.M.

1. Early Embryology of the Auditory Ossicles and Associated Structures in Relation to Certain Anomalies Observed Clinically

   Barry J. Anson, Ph.D. (Med.Sc.)

   Jerome S. Hanson, M.S.
   (by invitation)

   Shatik F. Richany, Ph.D.
   (by invitation)

2. Tissue Reaction Following Reconstruction of the Oval Window in Experimental Animals

   Richard J. Bellucci, M.D.

   Dorothy Wolff, Ph.D.
   (by invitation

3. Human Cochlear Responses to Sound Stimuli

   John L. Bordley, M.D.

   Robert J. Ruben, A.B.
   (by invitation)

   George R. Hager, M.D.
   (by invitation)

4. Some Physiological Factors in Inner Ear Deafness

   Merle Lawrence, Ph.D.

5. Distribution of Hearing Loss in Various Population Centers
Aram Glorig, M.D.

SUNDAY AFTERNOON, MARCH 13, 1960

2:00 P.M.

6. The Management of Middle Ear Lesions Simulating Otosclerosis
Francis A. Sooy, M.D.
(by invitation)

7. Experiences with the Stapedectomy Technique of Shea for the Surgical Treatment of Otosclerosis
David Myers, M.D.
Woodrow D. Schlosser, M.D.
(by invitation)
Richard A. Winchester, Ph.D.
(by invitation)

8. Vein Plug Stapedoplasty
Clair M. Kos, M.D.

9. Partial Stapedectomy
J. V. Hough, M.D.
(by invitation)

10. Experimental and Clinical Experiences in Oval Window Surgery
Harold Schuknecht, M.D.

* * *

*Open discussion of last four papers*

* * *

MONDAY MORNING, MARCH 14, 1960

9 A.M.

11. Acceleration as a Means of Determining the Sensitivity of the Components of the Non-Auditory Membranous Labyrinth
Walter H. Johnson, Ph.D.
(by invitation)

J. Brydon Smith, M.D.
Joseph A. Sullivan, M.D.

12. Localization of Monaurally Impaired Listeners
Richard Vichveg, M.A.
(by invitation)

13. Deafness Due to Mumps: Inner Ear Pathology
John R. Lindsay, M.D.
Patricia R. Davey, FRCS (Ed)
(by invitation

14. Action of Enzymes on Effusions Obtained from the Middle Ear
Carl F. Gessert, Ph.D.
(by invitation)
Elizabeth S. Baumann
(by invitation)
Ben Senturia, M.D.

15. Tumors of the Temporal Bone in their Reciprocal Behavior
George Kelemen, M.D.

MONDAY AFTERNOON, MARCH 14, 1960

16. Labyrinthectomy
Terence Cawthorne, FRCS

17. The Lurking Latent Cholesteatoma
Victor Goodhill, M.D.

* * *

*The Profession is cordially invited to attend.*

## NATIONAL INDEX ON DEAFNESS, SPEECH AND HEARING

Gallaudet College and the American Speech and Hearing Association have established a National Index on Deafness, Speech and Hearing. This Index will combine the present indexing and abstracting functions of the Central Index of Research on the Deaf at Gallaudet College and the projected indexing and abstracting functions of the American Speech and Hearing Association.

The purpose of the National Index is to index and abstract all professional literature pertaining to deafness, speech and hearing and to make this material readily available through a regular, professional publication.

Information may be obtained by writing to:

National Index on Deafness, Speech and Hearing

Gallaudet College

Washington 2, D. C.

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## CASSELBERRY PRIZE

A sufficient fund having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available in part or as a whole, for a prize award. Theses must be in the hands of the Secretary of the American Laryngological Association prior to January 1 of any given year.

The Award is a prize of money with accompanying certificate signed by the officers of the American Laryngological Association. The sum of money will be agreed upon by the Council of the Association after the manuscript has been evaluated by the Award Committee. It may be awarded in whole or in part among several contestants.

Eligible contestants may be: 1. Hospital interns, residents, or graduate students in Rhinology and Laryngology; 2. An individual with an M.D. degree who is actively practicing or teaching Rhinology and Laryngology in the Americas; 3. Any scientific worker in the field of Rhinology and Laryngology.

Manuscripts shall be presented to the Secretary of the Association under nom de plume which shall in no way indicate the author's identity. There shall also be a sealed envelope bearing the nom de plume and containing a card showing the name and address of the contestant which the Secretary shall keep in his possession.

Manuscripts must be limited to 5000 words and must be typewritten in double spacing on one side of the sheet. The thesis shall not have been published elsewhere before submission.

The successful thesis shall become the property of the American Laryngological Association but this provision shall in no way interfere with publication of the thesis in the Journal of the author's choice. Unsuccessful contributions will be returned promptly to their authors.

The Award which will be made at the Annual Meeting of the American Laryngological Association shall be based on:

1. Originality of material.
2. Scientific and clinical value.
3. Suitability for this Award.
4. Method of presentation as to style, illustrations and references.

The maximum amount of the Award shall not exceed $200.00.

Lyman G. Richards, Secy.

Massachusetts Institute of Technology

Cambridge 39, Mass.

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## ANNALS

In order to fulfill the requests for the March 1959 and June 1959 issues, the ANNALS will pay $3.50 for each book in good condition.

Please mail books to Zimmerman-Petty Linotyping Co., 2308 Olive, St. Louis 3, Mo.

# OFFICERS

OF THE

## NATIONAL AND INTERNATIONAL OTOLARYNGOLOGICAL SOCIETIES

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### American Academy of Ophthalmology and Otolaryngology

President: John H. Dunnington, M.D., New York
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, October, 1960.

### American Board of Otolaryngology

President: Bernard J. McMahon, M.D., St. Louis
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa
Examination: Chicago, October 1959

### American Broncho-Esophagological Association

President: Daniel C. Baker, Jr., M.D., 903 Park Avenue, New York City, N. Y.
Secretary: F. Johnson Putney, M.D., 1712 Locust Street, Philadelphia 3, Pa.
Meeting: Deauville Hotel, Miami Beach, Florida, March 15 and 16, 1960

### American Laryngological Association

President: W. J. McNally, M.D., Montreal, Canada
Secretary: Lyman G. Richards, Mass. Inst. Tech., Cambridge, Mass.
Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960

### American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Theo. E. Walsh, St. Louis
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N.Y.
Meeting: Deauville Hotel, Miami, Fla., March 15, 16, 17, 1960

### American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Paul H. Holinger, M.D., Chicago, Ill.
Secretary: Walter E. Heck, M.D., San Francisco, Calif.
Meeting: Miami Beach, Fla., June 13-17, 1960.

### American Otological Society

President: Robert C. Martin, M.D., San Francisco

Secretary-Treasurer: Lawrence R. Boies, M.D., University of Minnesota Hospitals, Minneapolis 14, Minnesota

Meeting: Deauville Hotel, Miami Beach, Fla., March 13-14, 1960

### VII International Congress of Otolaryngology

President: Prof. M. Aubry, Paris

Sec. Gen.: Dr. H. Guillon, 6 Ave. Mac-Mahon, Paris XVII France

Meeting: Paris, July 15-22, 1961

### International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

### Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose Gros, Havana

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

### Pacific Coast Oto-Ophthalmological Society

President: John F. Tolan, M.D., 1118 - 9th Avenue, Seattle 1, Wash.

Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah

### The Society of Military Otolaryngologists

President: Lt. Colonel Stanley H. Bear, USAF (MC)

Secretary-Treasurer: Captain Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, California

### Canadian Otolaryngological Society

President: Dr. Gordon H. Francis

Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia

Meeting: Jasper Park Lodge, Jasper National Park, Alberta, June 10-12, 1960

### Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana

Secretary: Dr. Carlos Valenzuela, Monterrey 47 Mexico 7, D.F.

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